BRAIN.

JULY, 1884.

Original Articles.

ON THE SENSORIAL LOCALISATIONS IN THE CORTEX CEREBRI.[1]

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WHOEVER has made experiments on the sensorial centres knows how great and how many are the difficulties there are in proving the existence of disorders, total or partial, unilateral or bilateral, of the several senses. There is in this kind of research a constant danger of confusing objective facts with the individual appreciation of them. I therefore took pains to render more secure and rapid the diagnosis of sensorial alterations in animals whose brains have been operated upon, by making a comparative study of the value of the various methods employed, and finding new means better adapted to my object. I shall limit myself here to a sketch of the most general method of investigation applicable to the safe and

[1] This paper contains an abstract, or preventive communication, of the chief results obtained by the author in a recent series of long and laborious experimental researches on the sensorial localisations in the brain of the dog and the monkey. The full memoir is to be published shortly in Italy. The present abstract has been translated by myself from Prof. Luciani's original Italian manuscript.—The reader will find abstracts of Luciani and Tamburini's previous researches in 'BRAIN,' Vol. I. p. 529; Vol. II. pp. 234 and 580; a criticism of Munk by Ferrier, Vol. I. p. 229; a paper by Ferrier on Amblyopia, Vol. III. p. 457; and an abstract of Ferrier's recent researches on Localisation in the present volume, p. 139.—A. DE WATTEVILLE.

rapid discovery of the chief forms of tactile, visual, auditory, and olfactory disturbances which present themselves in consequence of the extirpation of various cortical areas. This diagnostic method which, when employed methodically, in every case makes the results obtained perfectly comparable among themselves, consists in the observation and the definition of the behaviour of the animal under the various stimuli which act upon its several senses, during the time it is taking its food, with its eyes open or with its eyes bandaged, or again with one eye open and the other closed. One prepares for the animal some favourite article of food; for instance, boiled meat divided into small pieces, and when the hour for the meal has arrived, when the appetite of the animal is keenest, its favourite food is shown it and given it to taste; then its eyes are well closed by means of sticking-plaster. Now, especially if it be the first time, the animal will make with its leg a few efforts to remove it, but if a piece of meat is immediately thrown out to it it leaves the plaster alone, and occupies itself in a search for the food, sniffing vigorously with its nose close to the ground, and moving about with great rapidity. This is the most opportune condition for testing the tactile, auditory and olfactory senses: it is enough to touch the various points of the skin, on the limbs, head or trunk with a straw or a pellet of cotton wool fixed to the end of a small rod, that the animal should under normal tactile conditions suddenly react, quickly turning the head towards the part touched, whilst it goes on seizing the food; it remains passive, or almost so, however, if the points touched are the seat of the diminution of the general sensibility. This mode of testing the sense of touch gives such clear results as to leave nothing to be desired.

By throwing pieces of meat at intervals upon the plate, sounds are produced more or less perceptible according to the size of the pieces and the force with which they are projected. The animal then has not only the sense of smell, but also that of hearing to divert its search. As soon as it has swallowed a piece of meat it stands motionless, pricking its ears in order to catch the direction of the sounds of other pieces falling, and if the hearing is perfect on both sides, it quickly turns either

to the left or to the right, according as the food is thrown nearer to one ear than the other; whereas if one of the two sides (or both) is deficient, the animal behaves in such a way as to show the side on which audition is imperfect (or manifests the bilaterality of the lesion). This is the best means of discovering even slight and unilateral alterations of hearing; whilst the test with the diapason (made to vibrate in the proximity of one or other ear), calls, loud and sudden noises are of use for investigating more profound auditory troubles.

This method of testing by the mode of alimentation with closed eyes is also of use to appreciate, though less accurately, alterations in the olfactory sense. For this purpose, it is enough to eliminate the intervention of the sense of hearing, by first disposing on the ground pieces of meat at a certain distance from one another, and noticing the rapidity with which the animal finds them by olfaction only. Then, after obstructing one or the other nostril with a plug of cotton wool, and holding the animal in the arms, one gradually brings its nose towards a piece of meat; one can thus judge comparatively of the acuteness of the sense in the two nostrils by measuring the distance at which the meat lies, when the animal begins to sniff and stretch its nose. The rapidity with which it reacts to more or less powerful odours (ammonia, acetic acid, chloroform, turpentine, cloves, &c.) may fulfil the same purpose.

It is a pity that we cannot test with the same ease alterations in the sense of taste, which we have no other means of appreciating than the readiness with which the animal reacts to bitter substances (colocynth, quinine, digitaline, &c.) applied to the two sides of the tongue.

The careful examination of the visual sense is of the greatest importance, in order to secure a diagnosis of its disturbances, whether partial or total, bilateral or unilateral, such as hemiopia, amblyopia, psychical blindness, complete and absolute blindness. The best method here is still the test by alimentation, whilst one of the eyes is closed or both are open. Thus, for instance, after the left eye has been closed, small pieces of meat are thrown to the animal (avoiding the production of any sound) first in a line with its nose, or a little to the left, so that

the images be thrown upon the external segment of the right retina; secondly, to the right, so that the images be thrown upon the internal half of the same retina. The animal turns at once to seize its prey if the section of the retina on which the image falls is normal, but shows that it does not perceive it, if that part of the retina be blind; or finally shows that it perceives, but directs itself towards the food with some hesitation, and not in the precise direction if the retina be amblyopic. It is, in general, difficult to distinguish between hemiopia and hemi-amblyopia; more difficult to distinguish between amblyopic phenomena and those of psychical blindness —an expression by which we intend to designate, with Munk, a peculiar condition in which the animal preserves the capacity of seeing, but has lost that of recognising objects. In order to succeed in this diagnosis it is necessary to place on the ground small pieces of meat along with pieces of sugar, or some other inodorous and non-alimentary substance of the same size. If the animal is simply amblyopic, it has only to approach the pieces of sugar in order to recognise them by mere sight without smelling or tasting them; but if it is psychically blind, if it has lost the memory of visual signs, it requires more than sight to recognise them, viz., smell and taste. This test gives sufficiently clear results in dogs, very evident ones in monkeys.

In certain cases, dogs operated on at certain points of the cortex show themselves completely blind to food placed in small pieces on the ground; in order to find them they employ smell and touch only. Still, we have not to do here with complete and absolute blindness (cortical, as Munk calls it), but perhaps with a psychical blindness of an extreme type with or without amblyopia. For if the animal be led into the open air in a garden where it has been accustomed to stay, one sees it proceed straight along the paths, successfully avoiding the contact of hedges, walls, and other fixed obstacles which it comes across; whilst on the other hand, it constantly strikes against any unusual obstacle, such as a board, placed across the path which the animal is following.

II. After this short description of the methods of investigation applicable to the organs of nerve, I shall now give an

account of the most important results obtained by me. I shall speak first of the cortical centre of the sense of vision, which has been the principal object of my researches, because it is the one which lends itself to the most searching physiological analysis.

The first question which presents itself with reference to the visual centre or sphere refers to the localisation of the same in the dog and the monkey. My new experiments point to the necessary conclusion, that appreciable visual disturbances follow not only extirpation of the *occipital*, but also the *parietal*, *temporal*, and *frontal* lobes. According to some results, the complete or partial destruction of the *cornu ammonis* also produces marked disorders of vision. Analogous results induced Goltz to admit, that in dogs the visual centre is so diffused in the cortex, that there cannot be said to exist any localisation of it. But if we consider more closely the effects of decortications of the various lobes, it is found, that lesions of the *frontal* and *temporal* lobes give rise to transitory visual troubles which gradually diminish, and finally disappear altogether (or at least become inappreciable), after a few days or weeks. Similar lesions of the *occipital* and *parietal* lobes produce permanent results; a certain improvement takes place during the first few days after the operation, but they then persist unaltered for months, or during the whole time of the observation.

We have observed another fact which does not agree with the doctrine of Goltz. When the lesions in the frontal and temporal regions are of small extent, that is to say, comprise but a single segment, sufficiently circumscribed, of these lobes, they may give rise to no appreciable disorder of vision, whereas equally limited lesions in the occipito-parietal province constantly produce evident visual disturbances.

We shall not be able to interpret these facts, considered in their whole and in their particular, without appealing to the theory that the visual sphere—though chiefly concentrated in the occipito-parietal zone of the hemisphere—is not clearly circumscribed therein, but enters into relation with other centres, as it were "par engrenage," by means of direct anatomical prolongations.

The experimental results obtained on monkeys are in accordance with this mode of interpreting the facts which relate to the visual sphere of the dog. My new experiments confirm on the one hand the capital importance of the occipital lobes as centres of vision, as already demonstrated by Munk, whilst on the other they confirm the view, that these centres spread by irradiation into the parietal lobes, especially into the angular gyri, and their fold towards the parieto-occipital fissure, as was shown by Tamburini and myself, and by Ferrier and Yeo. My new researches prove in addition, that the isolated destruction of the angular gyri produce slight, and rather transitory, visual disturbances; and that the effects of destruction of the occipital lobes are aggravated then considerably, but not rendered permanent by a posterior decortication of the angular gyri.

In order that the analogy between the visual sphere of the dog and that of the monkey should be complete, one would have to demonstrate, that extensive decortication in the neighbourhood of the temporal lobe of the monkey gives rise to transitory but appreciable visual troubles. The facts we have elicited bear out the statement, that in the monkey also the visual centres present such connections with other centres, as are considered by us as the organic condition of the great psychological principle of the association of sensations and ideas.

The second question relative to the visual centres consists in the determination of the relations existing between the retinal field of each eye, and both their relative cortical centres. My recent experiments confirm the view that the fibres of the optic nerve of the dog undergo in the chiasma an incomplete decussation, as was seen by Tamburini and myself, and that the extirpation of one of the occipital lobes produces a bilateral homonymous hemiopia, as Munk was the first to prove clearly, though more extensive in the eye of the opposite side. From this fact, now recognised as constant, it is logically concluded, that each of the visual spheres has a direct relation with the more extensive internal segment of the retina on the opposite side (including about two-thirds of the retinal field), and with the less extensive external segment of the retina on

the same side (including about one-third of the retinal field). But it does not follow from the fact that the direct and crossed fibres of the optic nerves correspond to two distinct segments of the retinal area, that they be distributed to distinct sections of the visual sphere, nor that each definite point of the retina have a fixed and constant relation with definite points of that sphere. I have obtained three different series of results which do not allow me to accept this conclusion.

1. Bilateral homonymous hemiopia results not only from complete extirpation of one occipital lobe, but also from extensive decortications of one parietal, and also of one temporal lobe.

2. Partial bilateral extirpations in the province of the occipital lobe (visual sphere of Munk) never produce distinct phenomena of partial blindness, but visual troubles, more or less accentuated, equally or almost equally diffused over the several segments of the two retinæ.

3. The hemiopic effects consecutive to extensive unilateral extirpations in the occipito-temporal and parietal region of the vortex, and the visual troubles diffused throughout the whole retinal field which occur after circumscribed bilateral extirpations in the same region, are not permanent but transitory phenomena.

These three groups of facts are, on the one hand, destructive of the well-known theory of Munk of the projection of the several segments of the retina upon definite segments of the visual sphere; on the other, they prove that though the crossed optic fibres arrive from the internal, the direct from the external retinal segments, they do not preserve their relative position throughout their long course, and in both cases enter promiscuously into relation with the ganglion elements of their respective segments of the visual sphere. Fig. A represents schematically our cortical visual centre or sphere: the dark points, which greatly predominate in the great occipito-parietal area of the left hemisphere, indicate the relations which the optic fibres coming from the internal segment of the right retina bear to the cortical centre of vision; the light points, which are more thinly scattered over the same region, indicate the relations which the optic fibres

coming from the external segment of the left retina, bear to the same centre. It is easy to understand how, on this hypothesis, the three groups of facts first mentioned find a natural and adequate explanation.

The same theory applies to the visual centre of the monkey. The only well-demonstrated fact of Munk's (and which is confirmed by my recent researches) is the bilateral homonymous hemiopia which immediately follows the extirpation of an entire occipital lobe. But according to the doctrine of Munk this phenomena should be permanent, whilst my results show it to be temporary, and reproducible by repeated partial extirpations of the same lobe. This confirms the fact that the visual sphere of the monkey extends beyond the limits of the occipital lobe, and proves that each segment of the visual centre is related with the fibres of the crossed tract as much as with that of the direct fasciculus of the optic nerve—just as what we have shown to obtain in the dog.

The third problem to solve is that concerning the functional nature of the visual cortical centres. Are the visual *perceptions* only, or are also the simple visual *sensations*, localised therein? (taking these expressions in the definite sense assigned to them by the followers of Flourens). Or in other words, the hemiopic effects consequent on extirpation of the whole visual centre on one side, and the absolute blindness following their complete bilateral extirpation, are they definitive and permanent phenomena as Munk believes; or do they (as happens after incomplete uni- or bilateral extirpations) tend to become gradually attenuated, until they become reducible to simple psychical blindness?

My own researches, though still incomplete with reference to the determination of the definitive effects of total extirpation of our visual centres, are however more than sufficient to be opposed to the theory of cortical blindness, as much as to that of the absolute and permanent blindness which Munk believes to have observed in consequence of the complete bilateral extirpation of his visual sphere (circumscribed, as we know, to the two occipital lobes). The fact is that after the most extensive extirpation of the occipito-temporal area, which is far more extensive than the visual sphere of Munk,

absolute blindness does not persist beyond a few days, and may even fail to supervene at all, if the extirpation is made by successive operations. The place of absolute blindness (when it occurs) is taken by a complete psychical blindness, through which the animal seems blind to any food placed on the ground before it, but seems still capable of avoiding fixed obstacles met on the way, and to follow the path in a straight line. Later on, the psychical blindness becomes incomplete, but yet permanent; the animal begins again to make use of sight in the search for its food, but is not able to distinguish meat from sugar by its visual impressions only, and remains in that state without any ulterior improvement.

These and many other new results which will be consigned and discussed in my forthcoming monograph, demonstrate that the cortical visual centre is not the seat of sensations of perceptions only; its function is to elaborate psychically the visual sensations which take place in the mesencephali ganglia, and more especially in the corpora quadrigemina, which must rank high in this respect according to well-established scientific facts.

Experiments on the monkeys confirm and throw fresh light upon this theory. The definitive visual symptoms presented by my monkeys after extirpation of the occipital lobes only, or of these and the angular gyri together, characterise clearly the psychical blindness in its opposition to absolute blindness and to simple amblyopia, and leave nothing to be desired from this point of view. When some time has elapsed after the extirpation, their visual sensations become perfect again; they are able to see minute objects, what they want is the discernment of things and a right judgment concerning their properties and their nature; they are deficient, in a word, of visual perception. For instance, if small pieces of fig, mixed with pieces of sugar, are offered to them, they are incapable of choosing by sight only, but require to take the sugar in their hand and put it in their mouth in order to assure themselves that it is not an alimentary substance. These are phenomena of psychical incomplete blindness, because the mutilation of the centres of visual perception has been incomplete. If the theory of Munk has been correct, that

not only perceptions but also sensations take place in the visual sphere, our monkeys should have shown a deficiency not only in the former but also in the latter.

III. What we have said concerning the centre of vision greatly facilitates the comprehension of all which relates to the other sensorial centres; for one may expect *à priori*, and with much probability, that the latter are constructed on the same general plan as we have just seen obtain for the former.

With reference to the *auditory centre*, our experiments teach that no evident disorder of hearing follows extirpations of the frontal and occipital lobes in the dog. If, however, we remove the zone intervening between the temporal and occipital lobes, undoubted evidence of auditory disturbance is obtained, leading one to suspect that the limit of the cortical auditory centre merges superiorly into the portion of the visual sphere, which corresponds to the external segment of Munk. The effects of extirpations in the province of the parietal lobe confirm what was recognised by Ferrier, and afterwards by Tamburini and myself, that the bend of the third external convolution certainly makes part of the auditory sphere in the dog, but demonstrate also that this sphere irradiates into the parietal zone above this convolution, into the region corresponding to the zone F of Munk.[1] The fronto-parietal tract also, which extends in front, above and below the fissure of Sylvius, and comprises portions of the 4th, 3rd, and 2nd external convolutions, and the lower and anterior portion of the gyrus hippocampi, certainly contain an important portion of the auditory centre; and their destruction, we find, produces marked disturbances of hearing. Our results tend to show that not only the whole cortical area of the temporal lobe, as admitted by Munk, but probably also the cornu ammonis (which anatomists consider as an introflexion of the cortex) forms an integral portion of the auditory sphere. Thus we may conclude that the auditory area irradiates from its central point in the temporal lobe, upwards towards the parietal region, forward towards the frontal, backwards toward the hippocampal, inward towards the cornu ammonis.

[1] See illustration representing Munk's Localisations, 'Brain,' Vol. II. p. 236; and Ross, 'Diseases of the Nervous System,' 2nd ed. vol. ii. p. 356.

With reference to the question as to the relation of the two cortical centres to the peripheral organ of hearing, my new experiments confirm more fully what Tamburini and myself had already recognised; each ear having connections with both auditory spheres, but chiefly with that of the opposite side. In fact, every unilateral extirpation of sufficient extent in the province of the auditory sphere causes a bilateral disorder of hearing, more marked on the opposite side; and every extirpation in the same province made on the opposite side, after the effects of the first operation have ceased to be apparent, does not only set up disturbances in the other ear, but reproduces also those which had disappeared from the ear on the same side.

These phenomena are not explainable otherwise than by admitting that the same conditions obtain in the case of the acoustic, as do in that of the optic nerve. In both we must distinguish a crossed and a direct fasciculus; the former consisted of a much larger number of fibres than the latter. Neither of these fasciculi possess any uniform relation with distinct segments of their respective cortical spheres, but their fibres irradiate themselves throughout the area of those centres. The schematic figure B represents the extension assumed by the auditory centres on the external aspect of the hemispheres; and the relationship of the crossed and direct fasciculi of the acoustic nerve to the cortex is indicated by the dark and light dots respectively. A comparison between the figures A and B shows how, from the superposition of the visual and auditory spheres, there results an intimate connection and union between the two, embodied in an intermediary zone in which the "engrenage," or working into one another of the two centres, takes place.

There now remains the question of the functional nature of the auditory sphere; we must determine whether this centre be destined, like that of vision, to *perceptive* action only, or whether it be one of acoustic *sensation* also, as Munk affirms. The effects of more or less extensive unilateral extirpations within the limits of the auditory sphere last, according to our recent results, for a longer or shorter time according to amount of cortical matter removed, but always disappear after some

time, or at least cannot be discovered by the means at our disposal. The symptoms consist in a more or less profound blunting of the acuity of hearing, but which never reaches complete deafness, and gradually fades off. An incomplete psychical deafness only subsists, by which the animal seems not to appreciate the meaning of sounds, noises, or calls, though it appears to hear them. The effects of a bilateral extirpation are always more serious: at first the auditory troubles may amount to absolute deafness, but soon pass into a condition of obtuseness of hearing; this also becomes attenuated and leaves behind it only more or less evident signs (according to the extent of the injuries) of psychical deafness. It seems therefore that Munk, in admitting a cortical deafness as a consequence of the bilateral extirpation of his spheres, is fallen into the same error which we alluded to with reference to the visual sphere, of attributing not only perceptive but also sensitive functions to the cortical centres.

IV. My new experiments on the *olfactory* centre or sphere are much less complete; but still, by collating those in which disturbances of the sense of smell were observed, we may gather some notion concerning the position and extent of the corresponding cortical area.

Extirpation of the occipital lobe never gives rise to disorders of olfaction; destruction of the temporal lobe, or rather of the zone F of Munk, on the contrary, is followed by such. This fact shows that this region contains considerable prolongations of the olfactory centre. No evident deficiency of smell follows the extirpation of the temporal lobe; but if the lesion extends to the neighbouring convolution above the fissure of Sylvius one observes a notable diminution of that sense. Finally, a number of experiments show that a more or less extensive decortication of the gyrus hippocampi, as well as a partial ablation of the cornu ammonis produce olfactory disorders, at first an almost complete loss of smell—a fact which seems to indicate to us that this portion of the brain is the central point of the olfactory sphere. Some of my experiments show, in addition, that the more or less extensive destruction of the gyrus hippocampi, as well as the partial ablation of the cornu ammonis, produces evident troubles, and

chiefly an almost absolute loss of olfaction, a fact which seems to show us that we have here the true centre, or fundamental nucleus, of the olfactory sphere.

These experiments, besides proving the great importance of the cornu ammonis in this respect, seem to point to a partial inter-hemispheric decussation of the olfactory fibres, analogous to what we said of the acoustic nerve—with this difference, however, that an opposite condition would prevail here, the direct appearing to be larger than the crossed fasciculus. Fig. C gives a schematic representation of the olfactory sphere, according to these views ; but it shows only the part of it which emerges from the base of the hemisphere (hippocampal region) towards its external aspect. The region extends from below the Sylvian fissure upwards and forwards, and spreads out as far as the parietal lobe of the hemisphere, and to a less degree towards the frontal lobe. The dark dots, which are more numerous, indicate the relations of the direct fibres with the cortex ; the light dots, present in smaller number, those of the crossed fibres.

We cannot state much concerning the localisation of the question for the sense of taste. The insufficiency of the means of testing it in animals, and possibly the hidden position they occupy on the cortex, may explain this want of success. Among our numerous researches we only once succeeded in proving a disordered condition of taste. After a unilateral extirpation of the fourth external convolution, and of a portion of the gyrus hippocampi, we discovered a diminished sensibility to the taste of bitters (powder of digitaline) on the half of the tongue corresponding to the lesion, as compared with the opposite half. From this fact we can only draw the presumption, that the gustatory is to be found in close relationship with the olfactory centre. And in fact, in the same dog, we were able to note the deepest alteration of the sense of smell.

V. My studies concerning the localisation of the *tactile* sense, or in other words concerning the *general sensorial sphere*, though still unpublished, were made in greater part in conjunction with Dr. G. Seppili, now physician-in-chief of the Asylum at Imola. Our results may easily be resumed as follows : consequently to the total or partial extirpation of the

motor zone in the dog and the monkey, one constantly observes effects not only of motor paralysis, but also of cutaneous and muscular anæsthesia. When the lesion is not very extensive, one may, within a few days, obtain an almost perfect compensation of the paralysis of motion and sense; but when it involves the whole motor zone, or even if it is deep enough to reach the opto-striate tract, the phenomena are persistent, and one may have clear evidence of them (though less marked than at first) several months after the operation. After decortication of any one region which responds to electrical excitation with reactions circumscribed to a single part of the body, the sensori-motor paralytical effects are not limited to that part, but are diffused more or less to other parts of the body on the same side. Thus after extirpation of the motor zone of the posterior limb, there is a diffusion of the paralytical effects into the anterior limb, and *vice versâ;* after extirpation of the excitable zone of the anterior limb and face, or of the face only, or of the neck and trunk, there is diffusion throughout the same half of the body. We have already asserted, that these effects are more intense in that portion of the body of which the cortical area has been chiefly or solely removed. Thus, to quote but one instance, after decortication of the excitable area of the anterior limb only, the paralytical phenomena, though they extend into the posterior limb, are mainly localised in the anterior.

From these results we must necessarily conclude, that a localisation or precise delimitation of single centres in the sensory-motor zone, based upon not their reactions to electrical excitation of various points of the area, as Hitzig and Ferrier were the first to do, but upon the effects of partial extirpations, as Munk pretends,—that such a localisation is not practicable. We are led to believe that this is due to the fact, that the single centres in the sensory-motor zone are so completely bound up with, and, so to speak, let into, one another, that it is not possible to divide them with a clear and definite line, such as is the case when the cortex is incised and removed; so that in destroying a centre one necessarily eliminates a portion of the neighbouring centres.

The almost perfect compensation of paralytical effects con-

secutive to the extirpation of a single excitable area, and which takes place after a few days, even in the part of the body corresponding to it, is a fact which argues in favour of the partial overlapping of the special centres contained in the general sensorial sphere. We must not forget the fact, demonstrated by myself in previous papers, that the centres of the limbs in the dog are not limited to the external cortex of the sigmoid gyrus, but extend over the whole still wider surface of the sulcus cruciatus, and it renders very difficult the complete extirpation of a single one of these centres. Considering the general sensory sphere as a whole, there remains the question whether it is limited to the frontal lobe, or whether it spreads out more or less into the other cerebral lobes. My recent researches exclude the occipital and temporo-sphenoidal lobes, but show that it does extend into the parietal lobe, extirpations of which constantly give rise to more or less extensive and durable tactile paralyses or pareses.

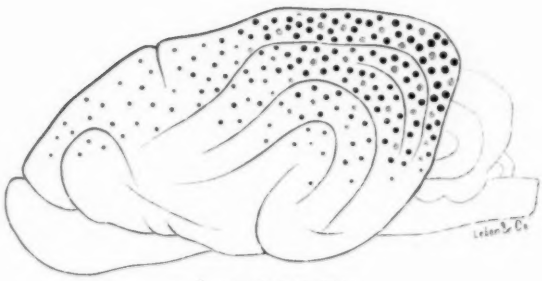
Let us remark finally, that in all experiments upon the tactile sphere there was a manifest and constant crossing of the relations between the peripheral sensory fibres and their respective cortical centres. Fig. D represents schematically the probable extension of the general sensorial sphere on the external aspect of the brain of the dog, comprising the several sensory-motor centres for the various parts of the body. One sees that what one calls "motor zone" is the central focus of the large portion of the sensorial sphere visible on the external aspect of the hemisphere—focus from which the latter is irradiated forwards towards the tip of the frontal lobe, backwards towards the parietal lobe, entering thus into intimate connection by "engrenage" with the visual, auditory, and olfactory spheres.

VI. If we compare together the four figures which indicate the localisation of these centres in the cerebral cortex, and ideally superpose them upon one another, we discover a remarkable fact, which I consider as one of the newest and most important results of my new inquiry. The four sensorial spheres, besides possessing each a territory of their own in the cortical substance, have, in addition, a common territory which is represented by the area of the parietal lobe, or more

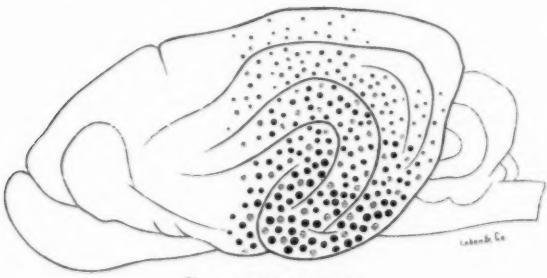
precisely by the zone F of Munk, which the latter calls the region of the eyes, attributing to it the humble functions of providing for the tactile sensibility of the eyeballs and conjunctiva.

In this region the mutual overlapping, or "engrenage," and consequently the partial fusion takes place of the single sensorial centres (excepting the gustatory centre, of which we know but little). Every one will thus see how this region is the most important in the hemisphere of the dog, where it represents, so to speak, the centre of centres. And indeed extirpation of this region, whilst it affects more particularly the visual perceptions, injures at the same time the auditory, olfactory, and tactile perceptions. There is no other portion of the brain of the dog lesions of which are capable of giving rise to effects so complicated, and so liable to set up profound psychical disturbances in the animal.

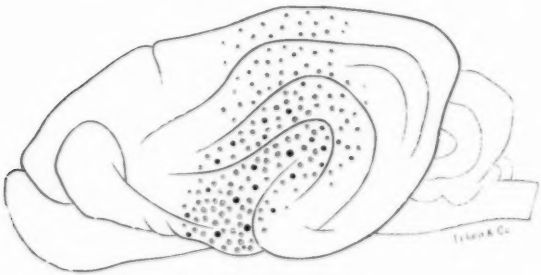
And in fact, after extirpation of the parietal lobes, Goltz—this diligent and able observer, but who starts from a point of view totally opposed to ours—has noted a notable alteration in the character of the animal; good-tempered and affectionate before the operation, they become peevish, quarrelsome, and refractory. He gives no explanation for this fact (which I have confirmed in a dog), and could not possibly give it on the grounds of his well-known theory. To me, on the other hand, this deep alteration in the psychical characteristics, appears to be referable to the loss of this important cortical area, in which the several sensory spheres meet and coalesce, and the removal of which necessarily produces the loss of the normal association of the psychical perceptions and images.



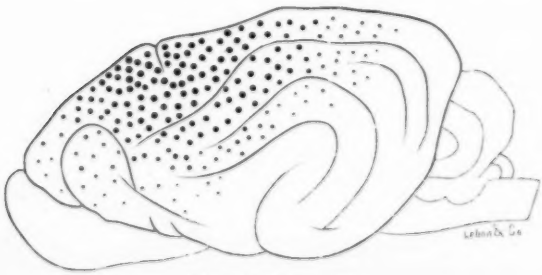
A.—VISUAL SPHERE.



B.—AUDITORY SPHERE.



C.—OLFACTORY SPHERE.



D.—TACTILE SPHERE.

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THE RELATIONS OF HYSTERIA WITH THE SCROFULOUS AND THE TUBERCULAR DIATHESIS.

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(*Concluded from* Vol. VII., *p.* 28.)

CASE XL. (condensed). (Observations of Raymond, 'Gaz. Médic.' 1881, p. 431.)—*Tubercular Heredity. Pulmonary tuberculisation. Hysteria beginning with exaggerated and obstinate sweatings.*

Heredity.—Sister died of phthisis.

History of patient.—Locksmith (25 years) is admitted for a bronchitis of three months' duration. Dulness at the apex of the right lung (infraspinous fossa). Expiration prolonged and jerky, and dry crepitation. Very abundant sweating during the night (not on the hands). Usual treatment of tuberculosis, and granules of atropia. After four days the generalised sweatings have disappeared, and their place has been taken by sudoral secretion of the palmar aspect of both hands; the secretion is very abundant, constant, and increased by the slightest emotion. Failure of atropia internally; transitory success when in local hypodermical injections; success more rapid and more permanent with Duboisia. However, the sweatings reappear, and resist a series of other treatments. The mind becomes impressionable, irritable, anxious. He is moved by nothings, cries easily, and presents various nervous phenomena. Epigastric constriction, strangulation, repeated hysterical attacks. Right hemiplegia and hemianæsthesia, with contractions, aphonia, etc. "The general health in the interval of the attacks is perfect."

This case has the great advantage of being recent, and of having been followed with the greatest care. It therefore establishes incontestably that in a patient hereditarily tuberculous, there have been, first of all, typical signs of the

pulmonary tuberculosis; that those lesions, instead of progressing (as it is the custom) have stopped, and probably even retrograded; and that, in their stead, a hysteria quite typical and complete has developed itself.

Let us notice, before passing on, the fits of sweating which appear here as in our patient in Case II., and which are useful to show, so to speak, the transition between the period of the pulmonary and that of the neurotic symptoms.

Case XLI. (condensed). (Brousse, 'Gaz. Hebd. of Montpellier,' 1881, p. 421.)—*Hereditary neuropathic antecedents. Personal scrofulous antecedents. Pulmonary Tuberculosis. Convulsive Hysteria.*

R., a soldier, admitted June 29, 1880, into the hospital St. Eloi.

Brother died of convulsions; sister has curious nervous disturbances at each menstrual period.

He looks delicate, rather effeminate; has shown in his youth evident signs of scrofula (scabs on the head, repeated blepharitis, etc.); when four years old violent measles, which leave after them a particular susceptibility of the respiratory apparatus. Since that time the patient is easily affected by bronchitis; he takes cold almost every winter, and at such times expectorates freely; expectorations often sanguineous.

Since ten years of age, convulsive attacks hysteriform in character: "First feeling of weakness in the lower limbs (he then knows that his attack is coming, and can save himself from a dangerous fall), then sensation of abnormal weight in the epigastric region, which from there goes upwards to the neck and seizes him at the throat; then beginning of the attack properly so called by a fall (without the cry), with loss of consciousness, afterwards clonic and sometimes tonic convulsive movements. After a few minutes the attack ends, the patient opens scared eyes, sometimes begins to cry. Soon after he is seized with a very acute feeling of fatigue, shuts his eyes and sleeps for many hours."

At his admission into the hospital, the sensibility of the whole body is noticed to be obtuse, with some hyperæsthesia, painful vertebral spots, reflex hyperexcitability, etc. In the chest signs of tuberculosis beginning in the apex of the right lung; a few dry crepitations, with harsh respiration and prolonged expiration in the right supraspinous fossa. In the heart there was a certain amount of stenosis of the pulmonary orifice.

Attack, during the visit of June the 30th. "Whilst he is being auscultated, R. suddenly gets pale, loses consciousness, exerts him-

self as if he wanted to lift up a weight which, starting from the epigastrium, seems to rise up to the neck and to suffocate him; afterwards appear clonic movements in the limbs and especially in the fingers; at the same time movements of translation of the body to the right and to the left, convulsions of the eyes upwards and contractions of the pupils. After a few minutes the patient regains consciousness, but the look is scared; the pupils are very dilated; the patient feels exhausted, and soon falls into a deep state of sleepiness, without stertor. During attack there have been neither froth at the mouth, nor biting of the tongue, nor even turgescence of the face." Similar attacks occur repeatedly. Dysentery.

On the 15th of August, the pulmonary lesions noticed when he was admitted have made no noticeable progress.

On the 1st of September the patient is dismissed, and leaves the hospital in a better state of health than when he entered it, without however being completely cured.

In this case, we have no alternations between the thoracic and the neurotic symptoms. The two orders of diathesic manifestations exist together. Only the very slow progress of the tuberculosis is to be noticed, as well as the slightness of the pulmonary lesions, although the patient has been examined when 21, and that since 4 years of age (measles) he has had frequent bronchitis and spitting of blood.

Case XLII. (personal). (Condensed from the notes of MM. Brousse and Sarda.)—*Hysterical phenomena. Progressive Pulmonary Tuberculosis. Death and Autopsy.*

Virginie M., 47 years of age, admitted at the General Hospital the 28th of July, 1879.

Mother of a nervous temperament, died dropsical when 74. Rheumatic father. Nothing to note about brothers and sisters.

Courses appear when 17 years old. After six months disappear after a bath. At that time violent frontal cephalalgia, terebrant (like clavus); pains at the apex of the head; insomnia; fanciful appetite. Character sad, melancholic, sometimes excessively merry; frequent gastric cramps. Soon after, sensation of globus hystericus starting from the abdomen. The courses are re-established after five months. Paludean fevers repeatedly.

Married at 25, has nine labours or abortions. Frequent amenorrhœa, often intractable, brought on by emotions. After the death of one of her children (three days after birth), the nervous state

grows worse. More intense and more frequent cephalalgia as well as sensation of globus; menstrual disturbances. At that time, persistent spot (for 10 months) of painful anæsthesia on right side of the thorax. Treated in succession in various wards of the hospital St. Eloi, sometimes for a metritis, sometimes for hysterical phenomena (peritonism, insomnia, various neuralgiæ, profound depression, etc.).

When admitted into the hospital, persistent pains more or less vague and erratic in character; state of hystericism without well-defined symptoms. All these neuropathic phenomena resist every kind of antispasmodic treatment. At the same time, dry, nervous, intractable cough. In November 1879, dulness in the right apex, in front and in the back; in the same region, prolonged expiration and respiration, harsh, even tubular, specially in front. The pains continue, however, to be very bad.

On the 15th of December, moist crepitations under the right clavicle. Local temperatures, 37· 2 (C.) under the right clavicle; 36· 7 (C.) under the left clavicle; 37· 2 (C.) in both axillæ. Always insomnia and considerable neuropathic malaise.

On the 14th of January, 1880, the pulmonary lesion has made alarming progress. Sibilant rales in the left apex; tubular breathing, humid crepitations, and cavernulous rales under the right clavicle. Greenish and very abundant expectoration. General state, always bad; much pain. Thrush in the mouth.

February 1st.—Signs of cavities under the right clavicle. Prolonged expiration, and harsh inspiration (without rales) in the left apex.

5th.—Delirium; pains, and moaning.

8th.—General state grows from bad to worse. The subcrepitant rales invade both lungs, the pulse very frequent becomes filiform. Progressive dyspnœa. Bronchial and trachæal rales, profuse sweatings, etc. Died on the 10th.

Post-mortem.—Right pleura thick and adherent on the whole extent of the middle and upper lobes, cavity in the apex communicating with a series of smaller cavernules. Grey granulations in the lower lobe. Analogous lesions (very advanced too) in the left lung. Passing over the other details of the autopsy (made however very carefully), let us merely mention four cysts in the broad ligament.

In this patient the hysterical phenomena have not been so marked as in the other observations. However, there has certainly been a state of obstinate neuropathy, a true hystericism.

The pulmonary tuberculosis has begun late, after a long period of purely nervous phenomena. It has rapidly taken the first place, and without entirely displacing the painful neuropathic state, has killed the patient in a short time.

This is the opposite of the cases in which the hysteria was on the contrary very pronounced, whilst the phthisis followed a course of remarkable slowness.

CASE XLIII. (personal). (Condensed from the notes of M. Brousse.)—*Hystero-Epilepsy. Pulmonary Tuberculosis.*

Marie F., 55 years old, admitted into the General Hospital the 11th of March, 1880.

Father died of old age when 94, is said to have had in his life-time dartrous eruptions. Mother died of a pneumonia when 58. One of her brothers suffered from incontinence of urine during his young age.

She has had small-pox when 6 years old, and an erysipelas of the leg when 9. Often dartrous eruptions. The courses, which have begun when she was 22, have always been scarce and of short duration. Married; two children in good health. When about 40, had jaundice and lost her hair. Menopause at 48. (Frequent headache before the courses showed themselves; has been bled many a time.) Nervous temperament. Frequent headaches, vertigo before the attacks.

Beginning of the present disease (attacks) about 7 years ago. One evening after having quarrelled with her husband, she goes to bed, and during the night she has an attack, during which she passes her water, and strikes her husband. She has had many hæmoptyses, the last about 7 or 8 months ago.

During a previous sojourn at the hospital St. Eloi she had undergone an anti-syphilitic treatment. Hystero-epileptic attacks have been observed there, some of which disappeared by compression of the ovary. In her attacks, at the beginning of which she shouts loudly, she loses consciousness entirely, falls (sometimes she hurts herself in falling), foams, passes her water, etc.

At the present time she complains of numbness in the right side, especially in the right hand; she often pricks herself without feeling it; cannot work long with that hand, which is weaker than the other (22 with the dynamometer on the right, 25 on the left; she was not left-handed). Notable diminution of the sensibility (general and special) on the right side: the sight is even almost completely lost on that side. Frontal cephalalgia usual. A

blister brings back a certain amount of sensibility. (I need not insist here upon the details of this æsthesiogenic action, which I have studied elsewhere.)

The 22nd of March one finds in the chest, under the right clavicle, externally, very plain dry crepitations during inspiration, with a slight dulness at the same level. Expectorates the following day sanguineous mucous matters, even at times pure blood. (She has had similar expectorations for about 8 months, and sometimes without any cough.)

29*th*.—Right ovarian pain under pressure. Sub-dulness under the right clavicle. Slight humid crepitations in upper third of that side. From time to time, dry crepitations under the left side. Behind, and on the left, dry crepitations also from time to time. Behind and on the right, slight humid crepitations less marked than in front. Obscured respiration in the basis of that side. Expectoration fluid, yellowish, tinted with blood.

Besides the major attacks she sometimes suffers from vertigo. If she is going about, she is obliged to stop and to sit down; if she is doing something she is incapable of going on, not knowing any longer what she ought to do.

It is an open question whether it is hysteria or epilepsy that we have to deal with in this case. In putting together all the details of the case (the hemianæsthesia specially), I incline rather to look upon it as one of hystero-epilepsy, that is of epileptiform hysteria. This being granted, we have another example of the coincidence of thoracic and neurotic manifestations in the same subject. It is the neurosis which predominates; the pulmonary tuberculosis began later, remained in the background, and progressed very slowly.

Case XLIV. (Tartivel, 'Union Médic.' 1864, I. p. 98.)—*Tubercular Heredity. Hysteria (cough; anæsthesia). Pulmonary Phthisis. Gradual disappearance of the spasmodic character of the cough.*

L. M. C., florist, age 20.

Father died of phthisis when 22, "after an illness of 18 months which it is impossible to mistake from the information given by the patient: heat and cold, neglected catarrh, repeated hæmoptyses, loss of flesh, continual cough, abundant expectoration, marasmus, death." Mother, on the contrary, strong and robust.

Sudden beginning with a violent fit of coughing, which lasted many minutes, threatened to suffocate her, and greatly frightened

her. Was quiet for a few days. Then, after having been upset, new attack, new intermittence, and so on. At the same time abundant sweating in the palms of the hands. Cutaneous sensibility very much diminished on the left side, in the upper and lower limbs, on their external surface. Perversion of the capillary circulation: mottling of the extremities and hypothermy; sensation of dead fingers. Nothing is found in the chest, and the diagnosis is made of hysterical cough.

After a few months only the tubercular lesions appear in the left apex, and then the nervous phenomena improve gradually. After a period of fugacious, intermittent attacks, the thoracic phenomena entirely replace the neurotic. At that time, "on the right, in the whole height of the chest, respiration sounds very much weakened, incomplete, and mixed with rare disseminated, subcrepitant rales, more numerous at the base. No marked vocal resonance; weakening of the plenimetric resonance in the whole surface, especially in the lower third. On the right, harsh supplementary respiration, almost tubular. Expectoration rare, evidently purulent. These various symptoms are such, the author concludes, that it is impossible not to recognise now the tubercular infiltration of the left lung, with symptomatic hyperæmia."

Tartivel admits that doubts may be thrown upon the hysterical nature of that cough. However, the various characters of which he reminds the reader, and which he brings into connection with the sensory disturbances, seem clearly to indicate that the functional or neuropathic element has played a predominating part throughout the first portion of the history of the patient, whilst in the second phase, the anatomical or pulmonary element has come to the front and gradually displaced the former.

III. Summary and Interpretation of the Facts.

The 44 cases adduced, naturally fell under two categories. The first contained those (25 cases) in which the neurosis was the only manifestation of the tubercular diathesis; the second those in which both the nervous and pulmonary symptoms occurred either at the same time, or in succession.

The first group shows the filiation in the same family of tuberculosis and hysteria; the second allows in addition to study the order of succession of the two sets of symptoms, and their

mutual influence upon one another. This group may be thus subdivided. With reference to the order of succession, three cases have presented themselves; 1, hysteria was manifested first, pulmonary troubles later (Cases XXVI., XXIX., etc.); 2, the opposite order was observed (Cases XXX., XXXI., etc.); 3, the two orders of symptoms occurred simultaneously, or nearly so (Cases XXVII., XXVIII., etc.).

Leudet, in an important paper ('Comptes rendus de l'Ass. franc. pour l'Av. des Sci.,' 1877, p. 829), comes to a similar conclusion: "Hysteria may precede, accompany, or appear in the course of, tubercular phthisis." He has seen only two cases in which hysterical followed pulmonary symptoms; a greater number of such instances will be found in the present paper.

With reference to the mutual influence of the two orders of symptomatic manifestations, a certain number of our observations show clearly a distinct alternation, the pulmonary symptoms taking the place of nervous symptoms and vice versâ. This substitution of one group of phenomena for the other may take place once in the course of the disease; or may, as we have also seen, occur several times.

When both orders of phenomena have had a simultaneous origin, this alternation is not observed; their evolution is synchronous, but even then there is evidence of their reciprocal influence, the pulmonary phthisis, for instance, being remarkably slow.

The final mastery remains sometimes to the one, sometimes to the other; we have mentioned cases where the hysterical, others where the pulmonary manifestations, definitively and exclusively conquered.

These propositions are more comprehensive than the conclusions of Leudet, with which however they are not in contradiction. He says: "Convulsive hysteria most frequently ceases on the appearance or in the first period of tuberculosis; . . . . its pre-existence does not prevent the rapid development of phthisis, but usually the latter runs a protracted course, and with longer periods of remission."

Let us now discuss the interpretation given to the facts by other authors, and bring this paper to a close with an endeavour to establish our views on a clinical basis.

1. It must be borne in mind that we do not deal here with two particular groups of facts upon which authors have much insisted, but form no part of our subject matter, viz., the pseudo-phthisis of hysteria, and the hysteriform symptoms of cerebral tubercle (pseudo-hysteria of phthisis).

With reference to pseudo-phthisis, it is well known that the neurosis may itself give rise to hæmoptysis, which if accompanied with loss of flesh and bronchitis, simulates the existence of pulmonary tuberculisation. Certain authors (e.g., Huchard and Mossé, loc. cit.) tend to refer to this category of cases all the instances adduced of phthisis and hysteria coexisting in the same subject. We admit that some cases may be objected to on this ground; thus for instance, certain observations by Largaud are not free from suspicion, and we mentioned them under all reserve. But let us not exaggerate, nor build up a whole theory on such slender grounds.

After eliminating all doubtful cases, a large number remain in which the phthisis was perfectly manifest, and in some confirmed by autopsy. Further, we adduce cases in which hysteria and consumption occur in the same family, but in different members. Hence it must be admitted that, making all allowance for, and putting aside pseudo-phthisis, there are certain relations between hysteria and true pulmonary phthisis.

We must likewise guard against the pseudo-hysteria of phthisis. Leudet and others have shown, with reason, the necessity of distinguishing the nervous phenomena arising from chronic diseases, and phthisis in particular; and we have endeavoured to keep this distinction before our eyes. We have, on the one hand, insisted upon the oscillations between the two orders of manifestations; on the other, we have carefully excluded cases where cerebral tubercles occurred.

2. The next explanation of the facts is the theory of the simple coincidence—a negation, not a solution, of the problem.

Thus Brachet will never see in pulmonary phthisis a true complication of hysteria, and with him many authors who adhere to the view formulated quite recently by Debove[1]:

[1] 'Leçons cliniques et thérapeutiques sur la Tuberculose parasitaire,' Paris, 1884.

"Considering the great frequency of phthisis, there must frequently occur patients whose fathers or mothers, or some relation, have died tubercular, without there being in the fact more than a coincidence. In other words, phthisis is such a common complaint, that if one wished to demonstrate by statistics its relationship to any disease, one would find among the ascendants or descendants of a large number of patients proofs of one's thesis." We recognise the importance of this argument, against which the present paper is wholly directed.

If, as recent exaggerations and distortions of pathological facts have led many to think, tuberculosis is not hereditary, and be contagious only; if the family history loses its importance in presence of the influence of the surroundings and of the facilities the subject offers for the introduction of parasites—then the occurrence of one or more consumptives among the ascendants of a hysterical person is of no greater consequence than that of a cancerous individual among those of one suffering from trichinosis. If, on the other hand, genuine clinical experience is right in attributing a very fatal influence to heredity in tuberculosis, if the family history throws important light upon individual facts (as Bouchard showed with reference to diseases from a slackening of the nutritive processes), if clinical medicine can no more isolate the patient from his ascendants and descendants than from his actual surroundings, if diathesis be not an empty name but expresses an unfortunately too real element of disease—if all this is true, as we firmly believe, then the cases collected in this paper will exclude the possibility of a mere coincidence.

Quite recently I have had under my observation two families whose history might well have been recorded along with the above cases. In the first, two out of eight children died from phthisis; two have very frequent and violent sick headaches; one is a convulsive hysteric, another suffers from attacks of somnambulism. In the second, with marked tubercular heredity, three out of six children died consumptive, one is insane, another hysterical. Shall we see here mere coincidences, establish no relationship between these morbid manifestations, and attribute to fortuitous circumstances the fact that those who have escaped pulmonary disease are neurotic?

No; they all labour under the same diathesis, the manifestations of which are pulmonary or nervous. To speak of mere coincidences is to go against general pathology and clinical medicine.

I believe that every physician who will carefully look about him, not in hospital, but in private practice, will be brought like myself to recognise certain relationships between hysteria and phthisis unexplained by the above-mentioned theories.

3. A different interpretation has been given, by Bernutz and Jolly, of the influence of the tuberculosis of parents upon the hysteria of children. "A much more serious objection," says the former ('Dictionnaire' of Jaccoud, p. 196), "may be raised against the opinion of Chairon, that pulmonary phthisis among the ascendants may be a predisposing cause to hysteria among the children .... this neurosis is much more common among those who have early been left orphans .... it is by depriving them of their father or their mother that phthisis favours the development of the neurosis among the descendants." The circumstance alluded to in this passage could, however, be called in to explain the development of tubercles, as much as that of hysteria in children deprived early of their parents and exposed to ill-treatment. Among our cases we find eight where the information necessary to elucidate this point is not given. In five others (II., VII., XII., XIII., XIV.), the view advocated by Bernutz might find some support; but in ten others it is inadmissible. Thus in Cases I. and III. the father and mother are living, and the child belongs to a good social position and is well cared for. In Case IV. the social position is high, and the disease appears at the age of 20; likewise in Case V. it manifests itself after marriage. The patient in Case VI. has been well brought up by the father and grandmother; in Cases XV., XX., XXIV., the neurosis appears late; and both parents of the patients in Cases XVI. and XXIII. are living. We look therefore upon the theory of Bernutz as insufficiently supported by facts.

According to Jolly (Ziemssen's 'Cyclopedia,' vol. xii.), it is not the fact of being an orphan early in life, but the congenital debility, which produces hysteria among the children of phthisical parents. "Various enfeebled conditions of

parents, from chronic disorder or otherwise, tend to develop a hysterical disposition among the children. Such an influence has more particularly been attributed to phthisis."

We shall meet this theory with a diametrically opposite affirmation by Briquet: "Organic diseases of the heart, pulmonary consumption . . . . are not usually followed by hysterical symptoms, whatever pain and weakness they may cause . . . . hence we must conclude that a great perturbation upon which all the vital forces are concentrated, does not allow the development of the nervous orgasm in which hysteria takes its origin."

As a matter of fact, in families where the diathesis prevails, it is not those most debilitated by the hereditary taint who become hysterical, for they become consumptives of the common type. Those on the contrary who, at least temporarily, resist the pulmonary affection develope the neurosis.

4. In opposition to the theories which seek to explain hysteria by the antecedent tuberculosis, we have those which tend to connect the final tuberculosis to an anterior hysteria. Thus Bernutz (loc. cit., p. 274) admits that hysteria may favour "among predisposed subjects the development of certain chronic diseases, more particularly of consumption, which since F. Hoffmann has been generally considered as a possible (and I may say pretty frequent) consequence of the neurosis."

Briquet likewise says (loc. cit., p. 537): "It is easy to understand how Hoffmann, Louyer Villermay, and Georget, seeing the nutrition of hysterical people to be disturbed either through the want of a sufficient alimentation or through worry of constant pain, ascribed to this fact the production of tubercles. All authors, indeed, allow that phthisis may arise as a sequel of hysteria, and there are good reasons for adopting their opinion."

Other writers, however, have opposed this view. Thus Monneret and Fleury ('Comp. de Méd.' v. 79) say: "Tubercle has been considered as complications depending on hysteria. Its development may be favoured by nervous troubles impeding respiration, but it will be conceded that it is going too far to speak of phthisis as a result of hysteria." Brachet ('De l'Hystérie,' p. 383) also says: "Cheyne is mistaken

when he says that phthisis was the result of violent hysteria." Huchard (loc. cit., p. 1056), too, considers it an error to say that hysteria favours the development of tuberculosis.

Finally Leudet (loc. cit., p. 831) answers these theories by the following statistics: "I said that 95 hysterical women had been admitted several times into my wards at intervals varying from 2 to 22 years. Now out of these 95, only 9 presented symptoms of pulmonary consumption. Out of 324 hysterical patients, only 23 showed signs of consumption. Hence I may conclude that phthisis is not common among the hysterical, and that the neurosis does not constitute a predisposition to organic lung disease."

We personally shall answer to those who will see in phthisis a consequence of hysterical enfeeblement, that this neurosis is, on the contrary, remarkable for the preservation of strength and resistance, which is observed even in cases where alimentation and nutrition are in abeyance. Further this theory does not explain the facts in which hysterical symptoms appear at a later date than the thoracic lesions, and still less those wherein hysteria is the only representative in the individual of the hereditary tubercular diathesis.

5. Many authors have been chiefly struck by the compensatory oscillations of the neurotic and tubercular manifestations, and have interpreted them as an antagonism of two diseases lying alongside, and have framed their views accordingly. Briquet states that he has seen phthisis "suspend the progress, and remove every symptom of hysteria" (p. 509). Tartivel found the same in the case we have quoted, and repeats that "*febris spasmos solvit.*" Féré ('Arch. de Neurologie, 1882,' p. 170) reminds us of the cases of the 'Iconographie' where hysterical fits and anæsthesia disappeared during the last step of pulmonary phthisis, adding that "it is impossible to decide therefrom whether it is the general exhaustion which brings about the disposition of the nervous symptoms, if whether fever is enough for that purpose, or whether there is some other cause at work." He eliminates the first alternative by quoting a case where a simple pleurisy suspended the hysterical condition, and the second by quoting one where acute articular rheumatism did not have that effect.

Pidoux ('Etudes sur la Phthisie') has noted the moderating influence of hysteria upon tuberculosis. "Neuroses," says he (p. 151), "hysteria, melancholia, the proteiform neuropathic state known as "nevrosism," often act as moderating agents. Phthisis in neurotic subjects has a very slow development, and periods of long intermission. I have no doubt that such subjects, among whom the disease is not rare, offer to it an extraordinary and almost indefinite resistance; and in treating them, we must not pay too much attention to their abnormal nerves." And again: "If a pulmonary consumption is combined with a neurosis, the organic lesion will evolve very gradually; gastralgias and enteralgias, have often this effect. I look upon this statement as an axiom."

Leudet (p. 831) quotes the opinion of Briquet and that of Walshe, which he nearly adopts: "Walshe ('Diseases of the Lungs,' p. 476) says, with reason, that phthisis and hysteria appear as a rule to be antagonistic; in other words, the pre-existing hysteria disappears when the pulmonary disease takes a certain development. . . . The opinion of these two authors is true for most cases; still there are exceptions."

Finally, Largaud (Thesis, Montpellier, 1882) has, under the influence of his master, Dr. Cavaillon of Carpentras, clearly brought into relief the antagonism of hysteria and tuberculosis considered as two distinct affections. "We shall show how consumptives, even when most threatened, may live, thanks to hysteria . . . . the neurosis appears to play the part of a protector, as if in its presence the pulmonary complaint had its symptoms mitigated and its progress arrested." And again: "Hysteria in a consumptive is not a symptom of tuberculosis; it is not a complication; it is only a new morbid state, without causal relation with it. . . . The only thing which may be affirmed is that hysteria acts as a safety-valve. . . . This is another illustration in support of the ancient aphorism of the Father of Medicine, *Duobus Doloribus*, etc." And finally: "The chief conclusion of our thesis is that consumptives may, if not recover, at least live for a long time, thanks to their hysteria. . . We shall therefore respect the neurosis as affording an element of safety to those patients. . . ."

We grant most readily to these authors the fact, observed

by ourselves, of alternatives between pulmonary and neurotic manifestations; but we cannot conclude therefrom to a theory presenting the two states as distinct diseases, fortuitously coexisting in the same individual, and exercising a true antagonism upon one another.

First, if an alternation is common it is not constant, and we have given cases where no trace of antagonism could be discovered. Leudet also quotes cases showing that in the midst of hysteria, phthisis may make rapid progress instead of being arrested. All the facts observed by the authors mentioned in the two preceding paragraphs, and brought forward in support of the theory of a mutual pathogenic influence between hysteria and phthisis, are also opposed to the doctrine of their antagonism. The views against which we are writing on account of their narrowness and onesidedness, rest upon groups of well-observed facts, which are destructive of the conflicting generalisations drawn from them.

But in the second place, in order to admit the theory of their antagonism, we must assume that hysteria and tuberculosis are two complete and distinct diseases. Now, hysteria is a neurosis, and a neurosis is always symptomatic; it is a "syndromon," or collection of symptoms, and not a disease or morbid state. The theory, after all, rests upon the assumption of a fortuitous coincidence of hysterical with tubercular accidents; it excludes every hereditary filiation between the successive morbid manifestations, and is therefore liable to the objections raised in our second paragraph.

6. We may finally mention a last theory upheld by Huchard (loc. cit., p. 1073): "Among diathesic states pulmonary tuberculosis has been mentioned among the antecedents of hysteria. But it is only the rheumatic and arthritic diathesis which should be adduced as capable of giving rise to both tuberculosis and hysteria; the latter is in no way under the influence of the tubercular affection." We grant that arthritism may give rise to hysteria, perhaps even oftener than tuberculosis, but it is not a reason to attribute to it all the instances of the neurosis observed in tubercular families. If we analyse our cases from this point of view, we see that in the great majority it is impossible to find a proof to that effect.

We have reached the end of our critical review of the theories proposed, and are satisfied with none. Neither the pseudo-phthisis of the hysterical, nor the pseudo-hysteria of the tuberculous, nor a simple coincidence, can account for all the facts. We cannot say that the children of phthisical parents become hysterical on account of their inherited debility, or of their becoming orphans early in life; nor that hysteria predisposes to phthisis by debilitating the subject. We cannot admit of two antagonistic diseases, nor appeal to arthritism as a common source of the two orders of manifestations.

Upon the ground thus cleared remain standing the views held by the Montpellier School of Medicine, which we shall here briefly restate.

Tuberculosis, like all other diatheses, is an essentially general and constitutional disorder; pulmonary phthisis is but one of its manifestations. It may show itself as a neurosis, and more particularly as hysteria.

Just as chorea is often a rheumatic disease, angina pectoris a gouty disease, thus hysteria may be a tubercular disease, taking the word tubercular in its nosological, not in its anatomical sense. In speaking of hysteria as a tubercular disease, therefore, we do not assume the existence of tubercular matter in the nerve-centres, for we do not look upon tubercles as pathognomonic of the diathesis, which exists without them as they may exist without it.

The tubercular diathesis is essentially hereditary, but may be understood only if it be followed up in families under its various manifestations from generation to generation. We then see how hysteria may represent the diathesis in one of the members of the family as meningitis may do in another, or Pott's disease in a third.

Besides such cases (I.–XXV.), in which hysteria is the sole manifestation of the diathesis, there are others (XXV.–XLIV.) where the same individual presents simultaneously or successively the two orders of manifestations, pulmonary and neurotic. We then witness the typical phenomena which characterise other protean diseases: the two orders of symptoms alternate and replace one another; or if they be simultaneous,

influence and modify one another, until one of them gets the upper hand. In other words, the mutual relations between the phthisis and hysteria of the tubercular diathesis are like those between the cerebral and abdominal symptoms of typhoid fever, or between the visceral and articular manifestations of gout.

These remarks concerning hysteria and phthisis form but a fragment of a great and true chapter concerning the relationship of all neuroses and even of all nervous diseases to the diatheses and general diseases.

If we cannot understand the neuroses without perceiving their mutual relations, if the consideration of the "*neuropathic family*" is indispensable for securing a clear grasp of the phenomena of neuropathology, it is yet necessary to go still further and complete all this by the conception of the "*diathesic family*," outside of which we shall find nothing but isolated, scattered, meaningless units.

One word in conclusion on the therapeutic applications of our theory. Hitherto, the neurotic element of hysteria has much too exclusively attracted attention; the diathesic element has not been met by treatment. Though we have no specific against diatheses (except in the case of syphilis), we nevertheless have various rational means to deal with them. The treatment of rheumatism is not that of scrofula, nor that of tuberculosis. Hence, it is not indifferent to know whether a neurosis depends upon the one or the other constitutional state.

Three categories of indications will have to be fulfilled in the full treatment of hysteria. First, the actual symptoms are to be met with appropriate measures, such as ovarian compression, ether inhalations, æsthesiogenous applications, etc. Second, the state of the nervous system, which is to be modified at any cost: hydrotherapy is often useful; change of surroundings; electricity, galvanic, but more especially static. We have little faith in the bromides. Third, the diathesis forms the fundamental ground for a chronic exhibition of arsenic, sulphur, alkalies, chloride of gold, mineral waters, etc.

Unless these three orders of indications be duly taken into account, our measures, based upon a superficial diagnosis, must necessarily remain sterile.

ON THE CONDITION OF THE FUNDUS OCULI IN INSANE INDIVIDUALS.

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(*Continued from* Vol. VII. *p.* 50.)

General Paralysis of the Insane.

We have examined 66 cases coming under this designation; most of them have been examined more than once, and many several times; in all, 135 separate examinations have been made. Of these 66 cases, 49 were males and 17 females; the average age of the patients was 40·28 years.

Of the 66 cases, 43 (65·15 per cent.) we considered to be normal; 15 (22·72 per cent.) were decidedly abnormal, whilst in 8 others the changes were so slight that we did not like to pronounce them positively abnormal; if however these be added to the 15 distinctly abnormal ones, we get a percentage of 34·84 per cent., under this latter category.

A qualification is here requisite; in one of the cases (No. 37 in table), the changes noted were (mainly, at any rate) due to Glaucoma, and are therefore foreign to the issue before us; a similar remark applies to two others (Nos. 36 and 62) in which the abnormalities present were due in all probability to the associated retinitis pigmentosa; it would, we think, be safer to exclude these cases, which would therefore leave 12 distinctly abnormal, to which if we add the 8 doubtful ones we should get a percentage of 30·30.

PLATE I.

Fig. 1.

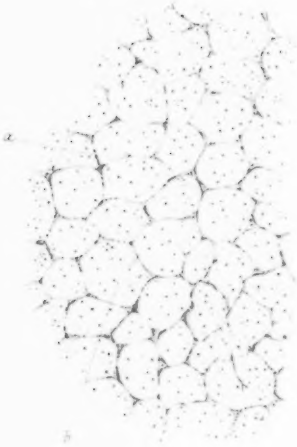


Fig. 3.

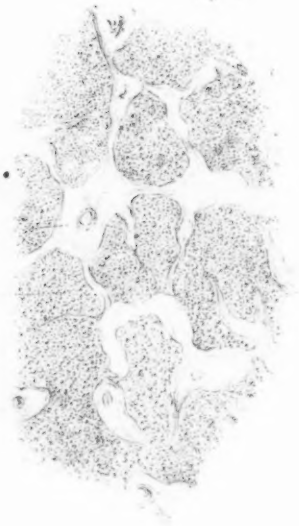
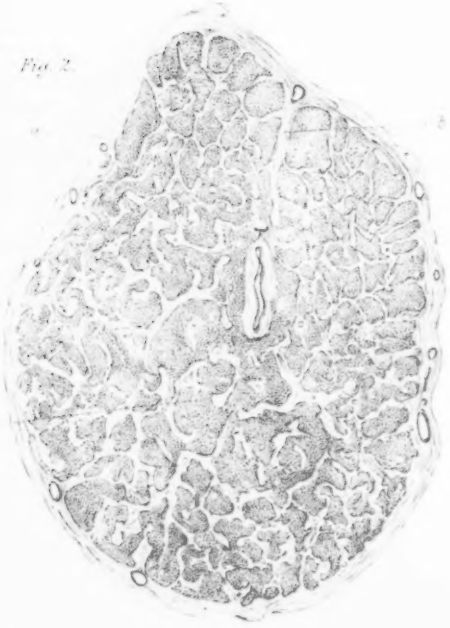


Fig. 2.



DESCRIPTION OF PLATES.

Plate I.

Fig. 1.—Transverse section of optic nerve, a short distance behind eyeball; from a case of General Paralysis of 3½ years' standing. (Case No. 23 in Table I.) Normal. × 50.

(*a.*) Normal trabeculæ.

(*b.*) Healthy nerve tubules.

Fig. 2.—Transverse section of optic nerve a short distance behind eyeball; from a case of General Paralysis. (Case No. 13 in Table I.) Atrophy; consecutive to slight chronic interstitial neuritis. × 50.

(*a.*) Thickened internal sheath.

(*b.*) Thickened trabeculæ.

(*c.*) Nerve tubules undergoing granular degeneration.

Fig. 3.—A portion of Fig. 2, × 300.

(*a.*) Greatly thickened trabeculæ.

(*b.*) Nerve tubules undergoing granular degeneration.

Plate II.

Fig. 4.—Transverse section of optic nerve a short distance behind eyeball; from a case of General Paralysis. (Case No. 28 in Table I.) Atrophy; primary at disc. × 50.

(*a.*) Greatly thickened internal sheath.

(*b.*) Thickened trabeculæ.

Fig. 5.—A portion of Fig. 4, × 300.

(*a.*) Thickened trabeculæ.

(*b.*) Neuroglia corpuscles, and small round cells.

(*c.*) Nerve tubules undergoing atrophy.

(The artist has here depicted the best part of the section; in most places the trabeculæ are thicker and closer together than are here represented.)

Fig. 6.—Transverse section of optic nerve, close behind eye-ball; from a case of General Paralysis. (Case No. 42 in Table I.) Early cirrhosis. × 300.

(*a.*) Thickened trabeculæ with well-marked nuclear elements.

(*b.*) Hypertrophied neuroglia corpuscles.

(*c.*) Healthy nerve tubules.

PLATE II.

Fig. 4.

Fig. 5.

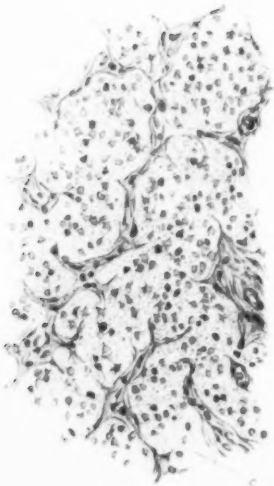
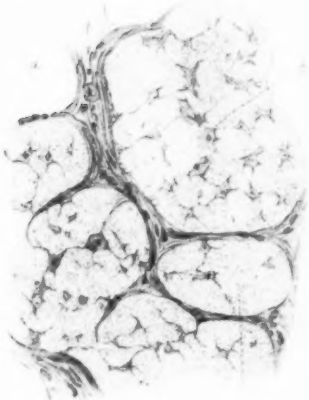


Fig. 6.



We append a table giving details of every case examined, and in the following account the numbers given refer to the numbers of the cases, in this table. (See pages 34 to 45.)

We have there divided the disease into stages, first, second, and third; the first being the early period of mental excitement and exaltation; the second dating from the subsidence of excitement, may perhaps be broadly characterised as one of advancing dementia, though at times patients improve very much at this period; whilst the third is the final stage of complete fatuity, during which the patient is quite helpless, and for the most part confined to bed. This is but a rough classification; the primary stage of exaltation is often absent; and there is no uniformity in any two cases, as regards the duration of the stages: nevertheless we have thought it well to include it, as giving at least a general idea of the period in each case at which the examinations were made. (It may perhaps be advisable to note that this division is not quite the same as that adopted by some authorities, who call the first stage, that of commencing mental alienation, prior to the advent of excitement, but the classification we have chosen we think preferable for the purpose.)

In 10 of the 66 cases the optic nerves and discs have been submitted to microscopical examination; the cases examined are indicated in the table, the details being given in the text.

It will be seen that four abnormal conditions of the optic discs have been met with. (1) Simple hyperæmia. (2) The same, combined with some amount of softening or blurring of the edges, the extent of this being never very great. (3) Simple anæmia. (4) Distinct atrophy. These are not however distinct conditions, but more or less different stages in one process, or we would rather say two, for we feel pretty confident that complete atrophy of the discs may occur without a previous stage of hyperæmia.

The cases in which decided hyperæmia of the discs occurred with more or less blurring of the edges are Nos. 3, 21, 34, 42, 13. In No. 3 the abnormal redness, &c., came on whilst the patient was under observation, and a microscopical examination of the parts involved was made after death, which took place two months after the last ophthalmoscopic examination.

In a transverse section of the optic nerve immediately behind the eyeball it was found that the trabeculæ, or fibrous septa between the nerve bundles, were thickened, in some parts decidedly so, in others scarcely at all: the neuroglia corpuscles were distinctly hypertrophied, sending out branching processes interlacing with each other; they also appeared more numerous than usual: these conditions were not uniform—some parts appearing healthy or nearly so. The nerve tubules seemed everywhere quite healthy. The conditions in fact were similar to, but less advanced than, those observed in case No. 42 (described below).

In No. 21 the discs when first examined were abnormally red, but this had subsided 2½ months afterwards, and these parts then appeared normal.

In No. 34 the discs remained in the hyperæmic condition during the six months patient was under observation, though the redness was subsiding when the last examination was made.

No. 42 was a good example of the process in question: the hyperæmia distinctly increased during the eight months the patient was under examination, and the edges of the discs were seen to become very misty and at length difficult to distinguish; the patient died two months after the last examination, of pneumonia, and a microscopical examination of the optic nerves and discs was made. It was found that the inner sheath of the nerve was scarcely at all thickened; but the trabeculæ were everywhere enlarged—in some parts very greatly so—the contained nuclei being very distinct; the neuroglia corpuscles were much hypertrophied, and their processes so numerous and distinct, that in many of the nerve bundles a distinct network of them could be traced with great facility; they appeared also to be increased in number. There was an absence of small cell infiltration in the nerve, but there appeared to be a little in the course of the fibres on the disc. The nerve tubules everywhere appeared quite healthy. (Plate II. Fig. 6.)

We wish particularly to refer to No. 13, which we think very interesting and important. This case was under observation 20½ months, and the process was watched through all

its stages, from commencing hyperæmia, to distinct atrophy. On the first examination, there was a suspicion of abnormal redness of the discs; on the second (two months later) distinct evidence of slight neuritis was found; this remained stationary for about a month, and indeed underwent very little change for another six months, though the redness seemed to be very slowly subsiding; at the end of 13½ months from the first examination, the right disc had become distinctly paler though the left continued red, whilst in another three months there was a still further diminution of colour in each disc; in another four months (20½ months from first examination) the right disc had become distinctly atrophic, whilst the left still retained colour, though this was fast subsiding.

We thus had here an opportunity of watching through its whole course a process which more often comes under observation at isolated stages, and though the inference that the process described occurred might have been legitimately drawn from the observance of the discs, at these isolated stages, the case just quoted furnishes an exemplification of this, as complete as could be desired. We should be disposed to style the process a slight neuritis,[1] running a very chronic course. A microscopical examination of the optic nerves was made in this case. The internal sheath of the nerves was moderately, but not greatly thickened; the trabeculæ were generally thickened, in some places very greatly so, and presented for the most part a translucent, imperfectly fibrillated appearance; in the centre of the thickest parts, enlarged arteries could in most cases be distinguished; the neuroglia corpuscles were increased in number, and to a certain extent in size, but there was no distinct small cell infiltration; in a few parts, what with the thickening of the neuroglia and the great development of the trabeculæ, the nerve bundles appeared almost obliterated, and in some of the sections a distinct band of fibrous tissue ran from the periphery inwards, in which but slight evidence of nerve bundles could be found; the fibrous thickening was thus much more advanced in some parts than in others. The nerve bundles appeared everywhere

[1] This term is perhaps not free from objection, but the process appears to be a chronic interstitial inflammation,—a cirrhosis, in fact.

to have undergone degeneration, their position being occupied by granular-looking masses, in which normal nerve tubules could not be distinguished. The nerves as a whole were much diminished in size. (Plate I., Figs. 2 and 3.)

It seems worthy of note that in two (Nos. 3 and 13) of these five cases in which evidence of slight neuritis was found, well-marked pachymeningitis of the cerebral dura mater extending quite down to base was disclosed at the autopsy; in two other cases however (Nos. 21 and 42) this condition did not exist; whilst in the other case (No. 31), no opportunity was afforded of ascertaining this point.

But whilst atrophy of the discs may thus succeed a process which can only be described as a slight chronic interstitial neuritis, we have also evidence that it may independently occur; but we must first notice a condition of anæmia of the discs of which Nos. 18 and 38 are examples. No. 18 was under observation for eight months; the pallor of the discs came on under observation, and was so great as to suggest atrophy; nevertheless this patient's acuteness of vision was perfect. We have before given it as our opinion, that if a patient can read J 1 and $\frac{20}{20}$ he has not as yet got atrophy, whatever he may be going to have. In the case before us, the pallor showed a tendency to diminish rather than to increase; we were not able to trace its subsequent progress owing to the patient's removal from the asylum, and hence, although the case may not improbably have ended as one of atrophy, we have no proof that it did so. No. 38 was another case in point, though here we had no opportunity of comparing the appearances presented by the discs with a previously normal state.

We have marked as distinct cases of atrophy, Nos. 28, 44, 45, 53, and 59.

In No. 45 the appearance of the right disc was strongly suggestive of commencing atrophy. This patient had the clinical signs of sclerosis of lateral columns of cord.

The other four cases were well-marked examples, and two of the patients (Nos. 28 and 59) were quite blind. No. 28 was a very interesting case. The patient apparently completely recovered from the first stage of the disease (as to the diagnosis of which there was at this time a little doubt), and

was discharged from the asylum; it happened that an ophthalmoscopic examination had been made, and nothing abnormal detected; nine and a-half months after the patient's discharge (about 14 months after the ophthalmoscopic examination) she was brought to the asylum by her husband, with the complaint that her eyesight was very bad, and had been failing since about a few weeks after her return home; both optic discs were then in an advanced condition of grey atrophy, and patient was nearly blind; her mental faculties were also clearly failing—she was in fact becoming demented. She was re-admitted into the asylum about 4 months subsequently, when the appearances presented by the optic discs were substantially the same, as they were also noted to be at the last examination, when patient was well advanced in the third stage of the disease.

A microscopical examination of the optic nerves and discs was made. It was found that the internal sheath of the nerve was very greatly thickened; the trabeculæ were also decidedly thickened and approximated to each other, forming in many places wavy bundles of fibrous tissue, the nuclear elements in these bundles being in many places very prominent. The neuroglia corpuscles were increased in number apparently, but not notably in size, whilst there was a general infiltration throughout the nerve bundles of small cells. The nerve bundles were much obscured by the thickened trabeculæ, and nuclear elements, and had clearly undergone considerable wasting, but could in most places be distinguished by careful examination. The nerve as a whole was much diminished in size. The transverse section above described, from this case, presented considerable difference as compared with that from No. 13; the chief points of contrast being that the internal sheath was more thickened, and the trabeculæ somewhat less so in the present case than in No. 13; the nerve bundles appeared also less degenerated in the former than in the latter, though possibly post-mortem changes may have been a factor in this; the nuclear elements throughout the section were also much more numerous in the case last described than in No. 13. (Plate II. Figs. 4 and 5.)

In No. 41 the atrophy, though not so extreme, was yet far

advanced. This case had the clinical signs of locomotor ataxy; and after death grey degeneration of the posterior columns of the cord was found throughout the greater portion of their length.

No. 53 is still under observation; the first examination was made when patient was yet in the first stage of the disease, and well-marked atrophy was noted then, without any trace of hyperæmia; the atrophic condition appears to have been steadily advancing.

No. 59 is also still in the asylum; he is quite blind, and complete atrophy of his discs was found when he was first examined; the patient was however at that time well advanced in the second stage of the disease, and the appearances of the discs were quite consistent with the former presence of neuritis; in fact they rather suggested such an origin.

This patient has well-marked signs of sclerosis of lateral columns of cord.

As regards the question of a relation between spinal symptoms and optic nerve atrophy in General Paralysis, our cases furnish both positive and negative information. Of the five cases of optic nerve atrophy which we have described, one (No. 44) had posterior sclerosis, and two others (Nos. 45 and 59) signs of lateral sclerosis. In No. 28, on the other hand, than which no case could have been more typical, not only were spinal symptoms absent during life, but from a careful microscopical examination of the cord, we are able to declare that it was perfectly healthy, sections taken from the cervical, dorsal, and lumbar regions, presenting perfectly normal characters. In No. 53, the knee jerks are normal, and there is no ankle-clonus. On the other hand, No. 66 has signs of lateral sclerosis, while the discs are described as normal.

We think that in at least three of these cases, Nos. 28, 44, and 53, the atrophy was primary, that is, was not preceded by any prior stage of hyperæmia at the disc, but the patients not having been under close observation previously, it is doubtless impossible to affirm this absolutely. It may, however, be remembered that in No. 28 a previous examination had been made, though sufficient time had probably elapsed between this examination and the second, for a neuritis to have set in

and run its course. In No. 53 again the discs were distinctly atrophic whilst the patient was yet in the first stage of the disease; in connection with which point it is not unimportant to observe, that those cases of slight neuritis which have come under our notice have run a very chronic course, remaining for months practically unchanged.

We ground our opinion, however, quite as much on the appearances presented by the discs at the time of examination, which were such as strongly to suggest that the changes were primary. The atrophy then would in all probability be due to destruction of nerve fibres by a chronic inflammatory process (similar to that we have noted at the discs in other cases) occurring higher up—in the intracranial course of the optic nerves—in the optic tracts—or even at the nuclei of origin; the nerve tubules below the seat of disease subsequently wasting.

In connection with this point it may be stated that in No. 28 the corpora geniculata had altogether disappeared, the corpora quadrigemina appearing normal to the naked eye.

The cases we have marked as doubtful are Nos. 1, 2, 22, 39, 43, 48, 51, and 60. We think it very possible that some at least of these cases were abnormal; in Nos. 39 and 60, for instance, the redness noted may not improbably have been the commencing hyperæmia of the neuritis previously described; whilst, on the other hand, in No. 43 the pallor may have been that of commencing atrophy; the changes being however so slight, we have preferred not to pronounce positively upon them. We may mention, however, that in No. 1 the blurring of the inner edges of the discs was probably a physiological condition, that was at least the impression conveyed by the appearance of the discs, which were perfectly normal in all other respects, and the condition in question has remained absolutely unchanged for 2¼ years.

It is unnecessary to comment upon the remaining cases which we have pronounced to be normal, except in the case of those, six in number, in which a microscopical examination of the optic nerves,[1] &c., was made after death.

No. 12. The last ophthalmoscopic examination was made

[1] Sections in all cases taken close behind the eye-ball.

2½ months before death, the patient being at the termination of the second stage of the disease, which had lasted about six years.

The nerve tubules were healthy; the trabeculæ were not thickened, but the nuclei in them were unusually distinct, and there was a very slight increase in the number of neuroglia corpuscles.

No. 23. The last ophthalmoscopic examination was made three weeks before death, the patient being in the third stage of the disease, which had lasted upwards of 3½ years.

The optic nerves were perfectly healthy. (Plate I. Fig 1.)

No. 24. Examination made two months before death, patient being in the third stage of the disease.

Optic nerves normal.

No. 27. Examination made a few hours before death, the patient being accidentally suffocated by food getting into his larynx, whilst yet in the second stage of the disease.

There was found a slight increase in the number of neuroglia corpuscles, with perhaps slight thickening of the septa here and there, but the nerves were otherwise normal.

No. 33. Examination one week before death.

Nerves for the most part quite normal, but in one or two places the trabeculæ appeared a trifle thick, and here also there was some slight increase in the number of neuroglia corpuscles.

No. 40. Examination made 3½ months before death.

Nerves for the most part perfectly normal, but septa perhaps a trifle thick in one or two places.

It will thus be seen that in some of the clinically normal cases there was found after death a tendency to increase on the part of the fibrous elements of the nerve, manifested by slight thickening of the septa, and slight increase in the number of the neuroglia corpuscles; the changes in question however were in all cases quite trivial.

By a perusal of the microscopical reports, particularly of course those of the abnormal cases, it will be seen that the pathological process is essentially one of increase in the fibrous elements of the nerve, at the expense of the nerve tubules; in cases Nos. 3 and 42, where the process was

examined in its early stages, a luxuriant overgrowth of the neuroglia elements was found, with thickening of the fibrous tissue framework, the nerve tubules being as yet perfectly healthy, whilst No. 13 presented us with a late condition in which the nervous elements had given way before the advance of the more lowly organised ones. Nor is it uninstructive to contrast these cases with those clinically normal ones, in which trivial changes were noted—changes however clearly tending in the same direction, though destined to remain in this very incipient stage; the changes indeed in these cases would very probably have remained in this incipient condition however long the patients had lived, though some might perhaps have advanced had sufficient time been allowed them.

In cases of primary atrophy such as No. 28, the pathological condition reached is eventually substantially similar, though here perhaps the sequence of changes may possibly be reversed at the distal extremity of the nerve.

As before mentioned, in the appended table details are given of all the cases examined. We do not think that there is any point in this, which calls for explanation, that has not before been commented on. As regards the refraction it will be seen, that there were eight cases of Hypermetropia and one of Hypermetropic Astigmatism; seven of Myopia, and two of Myopic Astigmatism; whilst the remaining 48 were either emmetropic, or the state of the refraction has not been recorded, in which latter case, the patients were in all probability emmetropic.

Though not strictly coming under the head of conditions of the fundus oculi, we have added a column for the state of the pupils, which have been recorded in nearly all the cases; we do so in order to emphasise the fact—not, we think, sufficiently known in this country—that there is a great tendency for the pupils in general paralysis to be inactive to light, whilst retaining their normal movement with accommodation—that is, to approach the Argyll-Robertson pupil, so common in locomotor ataxy. There is, however, as will be seen, much variation in this respect.[1]

[1] For a paper on the condition of the pupils, &c., in "General Paralysis," &c., see Bevan Lewis, in vol. iii. of Ophthalmological Society's Transactions.

Mania.—Taking all varieties of mania together, we have examined 82 cases: 6 of these cases were examined twice, and 1 three times, making a total of 90 separate examinations. Fifteen of these cases were males, and 67, females; the average age of the patients was 37·81 years.

Of these 82 cases, in 70 (85·36 per cent.) the optic discs and main retinal vessels were perfectly normal, whilst in 12 (14·63 per cent.) the condition of these parts was either abnormal or doubtful; that is, in these latter (doubtful) cases, the changes were so slight that no decided opinion as to their abnormality could be formed.

Taking the cases more in detail, we find that out of 34 described as "acute," 30 were normal, and 4 abnormal, or doubtful; out of 7 cases of "puerperal" mania, 2 were normal, and 5 abnormal or doubtful; whilst out of 26 cases of chronic mania, only 2 were described as abnormal or doubtful, and out of the 15 cases of the delusional form of mania only one, all the others being perfectly normal.

We append a table showing the condition of the optic discs, &c.; in these 12 abnormal or doubtful cases, upon which a few remarks may be made. (1) The changes are exceedingly trivial or inconsequential.

In No. 1, the discs were almost certainly normal; in No. 3, an old syphilitic choroiditis accounted for the majority of the changes noted; in No. 5, the only abnormality was that the veins were too full, but as in a subsequent examination this undue fulness had subsided, we have thought it advisable to include the case; in No. 10, the high degree of myopia was responsible for almost all the changes noted, though the appearance of atrophy in the right disc may have been independent. We have preferred to give these cases in full, in order that the reader may form his own conclusions upon them, but we are decidedly of opinion that some at least of the cases are normal.

2. It is, perhaps, worthy of note that almost all the changes noted, such as they are, are in the direction of abnormal redness of the disc; but in this connection we must not lose sight of the fact, that it is often as difficult to tell where the physiological limit of tint ends and the pathological begins, in the case of redness as in that of pallor.

3. The table brings out clearly the numerical preponderance of changes in the acute cases over the chronic.

Some further remarks are requisite with reference mainly to some of the normal cases. One of the cases was examined twice in the course of the acute attack, and another three times, but the discs presented precisely the same appearances at each examination.

We were able to secure a good view of the discs in 3 cases *during an acute maniacal paroxysm;* in one of these cases the discs were thought to be too red, and this case is included under the abnormal ones in the table (No. 9); in the 2 other cases the discs were noted to be perfectly normal; in one of these, however, it was only possible to make an indirect examination, whilst in the other the left disc only was examined, but a thoroughly good view of it was obtained by the direct method.

We have examined 3 cases shortly (1 to 3 or 4 days) after the subsidence of a maniacal paroxysm; in these the discs presented perfectly normal characters, no abnormal redness or pink suffusion being noted.

In 4 cases the condition of the discs was noted in the same patient, during the maniacal attack, and after convalescence; in one of these, which is included in the table of abnormal cases (No. 5), an undue fulness of veins was noted during the attack, which was not present when the patient was convalescent; in the 3 other cases the discs presented precisely the same appearances at each examination.

In a few of the 70 cases in which the optic discs and vessels were normal, some changes in the choroid were found. In one of these there was evidence of old choroiditis; in one, large posterior staphylomata, and in two others, small ones, due to the presence of myopia; whilst in one other small crescents were noted without any myopia; in another case again there was slight choroidal atrophy all round the discs, and in 3 others a little pigmentary disturbance of the choroid around or on one side.

In one case there was a detachment of the retina in the right eye. We have thought it well just to mention these points, but consider they have no bearing upon the subject of

our paper, for though we have included one or two such in our table of abnormal cases, this has been simply because the changes in the choroid were here associated with others in the discs, which latter were possibly independent of the former.

Melancholia.—In all, 49 melancholiacs have been examined, 8 of these were males, and 41 females. The average age of the patients was 38·93 years. In 40 of these cases (81·63 per cent.) the optic discs and main vessels were absolutely normal, whilst in the remaining 9 cases (18·36 per cent.) slight changes were found. More in detail; out of 7 cases of active recent melancholia, 5 were normal, and 2 abnormal or doubtful; out of 32 cases of the passive recent form, 27 were normal, and 5 abnormal or doubtful; whilst out of 10 cases of the passive variety which had become chronic, only 2 were in the latter category.

We consider that some of the changes noted are of very doubtful abnormality, but we have preferred (as in the case of mania) to give the cases in detail, in order that any one may be able to form his own opinions on the subject.

We may, however, remark concerning some of the cases in the table, that No. 2 was very probably quite normal; in No. 4, the only abnormality noted was that the vessels were rather small; in No. 3, the appearances of atrophy were most probably due to the patient's alcoholic excesses, which were almost certainly responsible for her insanity; whilst in No. 6, the changes noted were due to the presence of chronic Bright's disease.

We may just add, that in the 40 normal cases there were 2 in which choroidal atrophy was present round the discs, in one of which it took the form of a small crescent; in 2 other cases there were pigmentary deposits round the discs; whilst in yet 2 others a few small white spots were noted in the retina, due in one of these cases to chronic Bright's disease.

Mental stupor.—Two cases of mental stupor were examined, both during the attack and after convalescence; the condition of the discs is given in full in Table IV.; it would seem from the descriptions that the discs were perhaps slightly more pale during the condition of stupor than when the patients were

convalescent, but the difference was so slight that we think it would not be safe to draw any conclusions.

Dementia.—We have examined in all 61 cases of dementia, 7 of these were males, and 54 females; the average age of the patients was 46·80 years. Of these 61 cases, in 46 (75·40 per cent.) the optic discs, &c., were perfectly normal; whilst in the remaining 15 (24·59 per cent.) these parts presented some degree—either slight or considerable—of departure from the normal, the changes in some cases being so slight that no decided opinion as to their abnormality could be formed.

Examining the cases more in detail we find that out of 41 cases of dementia, secondary to mania, 33 were normal, and 8 were abnormal or doubtful; whilst out of 8 cases of primary dementia, 5 were in the former, and 3 in the latter category; of organic dementia again, out of 8 cases, 6 were normal, and 2 abnormal or doubtful; only 4 cases of senile dementia were examined, 2 of which were normal, and 2 the reverse.

As in previous cases, we append a table giving in detail the conditions noted in the 15 abnormal or doubtful cases.

It will be observed that in the majority of the cases given in the table, the changes were in the direction of abnormal redness; but the same remark applies here that we made in the case of mania, as to the difficulty there often is in defining the physiological limit of colour, and probably some of the cases included in the table were normal. In Nos. 7 and 12, the changes described were doubtless altogether due to the Bright's disease from which the patients suffered.

In 3 of the 46 normal cases, considerable posterior staphylomata were present, due to myopia; whilst in one other there was a small crescent without any abnormality of refraction being detected. In another case there was an irregular patch of choroiditis round each disc, whilst in 3 others there was more or less pigmentary disturbance of the choroid in this situation.

Epilepsy.—Of insanity associated with epilepsy we have examined 48 cases; 8 of these were males, and 40 females; the average age of the patients was 33·20 years.

Taking all the 48 cases together, in 41 (85·41 per cent.) the

optic discs and main retinal vessels were perfectly normal; whilst in 7 (14·58 per cent.) these parts were either abnormal or doubtful.

Of epilepsy associated with imbecility 20 cases were examined, 15 of which were normal, and 5 abnormal or doubtful. Of epilepsy, with slight impairment of the mental faculties, 20 cases were examined, only one of which showed any departure from the normal, and that very slight; whilst of the cases of epilepsy combined with considerable dementia, only one case out of 8 showed any tendency to abnormality, and here again the change was very trivial. As in other cases we append a table, showing the condition of the optic discs in the 7 abnormal (or doubtful) cases, from which it will be seen that the changes, such as they are, are very slight, and are chiefly in the direction of abnormal (or doubtfully abnormal) redness. Choroidal disturbances were noted in 5 of the 41 normal cases; 2 of these were merely myopic crescents; in one there was a zone of choroidal atrophy round the discs, and in one other some slight progressive choroiditis in the same situation, associated with chronic Bright's disease; while in yet another case there was slight pigmentary disturbance of the choroid around the discs.

It is perhaps worth a note, that out of the 48 cases no fewer than 20 presented errors of refraction; of these 12 were hypermetropic, and 2 had hypermetropic astigmatism; 2 were myopic, and the other 4 had myopic astigmatism.

A few observations were made with reference to the condition of the discs and retinal circulation before and after the occurrence of fits.

In one case a fit came on whilst the disc was under observation, but it was unfortunately impossible to keep this in view during the progress of the attack; immediately afterwards, however, the disc was thought to present a distinctly pink tinge, as compared with its condition just prior to the attack, when it was very pale.

One case was examined some hours after a series of 35 fits, but no abnormality was detected. In another case, a good view was obtained by the direct method immediately after a fit, which was one of a series of status epilepticus, comprising

something like 60; the disc was normal, of a red tint, with well-defined edges; the veins were full, but could not be described as abnormally distended; these characters remained constant during an observation of about 20 minutes.

We may mention in this connection, that in the case of a general paralytic (No. 33 in Table 1), who died after a series of convulsions, a view of the left disc was obtained by the indirect method just as a severe fit was passing off; the veins then appeared distinctly distended; a good view was subsequently obtained by the direct method and maintained for some minutes; the discs and vessels then appeared normal, though the blood in one of the main veins was perhaps a little darker than usual.

Imbecility.—Five cases of imbecility have been examined, *not* complicated with epilepsy; 3 of these were males, and 2 females; the average age of the patients was 22–80 years.

The discs were perfectly normal in every case.

Taking again the whole series of cases examined, one or two further remarks have to be made.

A point of some little interest which forced itself upon our attention, was the number of cataracts we came across; including all cases (slight and considerable) of opacities and striæ in the lenses, we noted that 43 out of the 313 cases were so affected—a proportion of 13·73 per cent. The average age of the patients was 54·72 years. We are not acquainted with any statistics bearing on this subject, but we should think that this was a much higher percentage than occurs in the population at large of the same average age. If this supposition be well founded, it would furnish another interesting exemplification of the general impairment of nutrition, so common in the insane.

We think it just worth mentioning, that out of the 313 cases we met with 4 in which the condition known as "opaque nerve fibres" existed in connection with the disc; the extent to which this occurred varied considerably in the 4 cases, and was in none of them very extensive; the condition, as is well known, has no pathological significance.

It is also perhaps worth a note, that in not a few cases we met with tortuosity of the vessels, this condition being extreme

in the person of one man suffering from the delusional form of insanity. We mention this as some observers appear to have attached weight to the circumstance; but in our own cases we feel confident that the condition was purely physiological, our experience furnishing us with numerous similar cases, in persons other than lunatics, in which it was not legitimate to invoke a pathological origin.

Only two cases of retinitis pigmentosa were met with, and both these occurred in the persons of female General Paralytics, and have already been referred to; in one case the changes were confined to one eye.

It would be foreign to the purpose of this communication to examine in detail all the previously published observations on this subject, even had we the material before us to enable us to do so. In Dr. Gowers' valuable work on the ophthalmoscope, references are given to some of the Continental publications on the subject, and the striking discrepancies between the observations of different observers is commented on. We will content ourselves therefore with some remarks on the observations which have been published in this country.

First then, as regards General Paralysis.

To Dr. Clifford Allbutt belongs the credit of being the first —in this country at least—to break ground on the subject, and his observations, first published in the 51st volume of the Medico-Chirurgical Transactions, are now to be found incorporated with his work on the ophthalmoscope.

Of 53 cases of General Paralysis examined, Dr. Allbutt only found 5 that were healthy; of the remaining 48 he found "atrophy of the optic discs in its various stages in 41 cases," and 7 cases were marked as doubtful; and the conclusion drawn was "that atrophy of the optic nerves takes place in almost every case of General Paralysis."

This conclusion is not in accordance with the results of our own observations.

On going carefully through the cases given in detail in Dr. Allbutt's tables, we find amongst the 41 cases, 4, in which pure hyperæmia was present, without the slightest appearance of atrophy whilst the case was under observation; in 14 others,

some amount of atrophy was combined with the appearances of congestion, or these latter had been noted at some previous examination; the remainder must therefore be intended for cases of pure atrophy. After a careful perusal of the descriptions however, we are unable to reconcile some of these with the conclusions drawn from them; at the least, we should say that some of them were of very doubtful abnormality. We note also that three cases of "Amaurosis" are given, without any mention being made of the appearance of the discs, which we therefore presume were not examined; we cannot, however, consider that to include these cases under the head of "atrophy" is altogether satisfactory, seeing how many misleading factors come into play in a demented General Paralytic, in the third stage of the disease.

We may also mention that in the case numbered 31 in Dr. Allbutt's tables, the atrophy of the left disc is at least as likely to have been due to the syphilitic choroiditis with which it was associated, as to have had an independent origin. Making every allowance for doubtful cases however, the percentage of abnormal cases in Dr. Allbutt's tables is very much higher than that which we have ourselves experienced. As regards the nature of the lesion however, in the abnormal cases, we are quite in harmony with Dr. Allbutt, the changes noted by us being clearly practically identical with those so well described by that observer.

In Vol. II. of the West Riding Asylum Reports (1872), will be found a paper by Mr. Aldridge, entitled "Ophthalmoscopic Observations in General Paralysis." Mr. Aldridge examined 43 cases, the details of which are given in his tables. Though the author does not distinctly state that all his cases were abnormal, he leaves it to be inferred that they were so; for on p. 225 of the work referred to, he states that "only two conditions of the optic nerve were observed," and then goes on to describe these conditions, as those of neuritis and atrophy; whilst on p. 227 he distinctly states that atrophy was present in greater or less degree in all the female patients (13 in number) that he examined.

Here again we note agreement as regards the nature of the change with the prior observations of Dr. Allbutt and those

now published by ourselves. The numerical proportion of abnormalities, however, detected by Mr. Aldridge, being so greatly in excess of anything we have observed, we cannot resist the suspicion that some error has crept in to vitiate his conclusions; nor indeed is internal evidence of this wanting; for on p. 226 of the work before us, we read: "The disease generally ends by one side of the disc, usually the inner, becoming white and atrophic, whilst the inflammatory changes are still in progress at the other. *In some cases again another course seems to have been taken, for in them we find that the disc has a white rim, and that a very large and shallow excavation of an extremely pearly white tint occupies the centre, the remaining portion being said to be of a greyish-pink tinge.*"

The italics are our own, and we emphasise this passage because we recognize in it a good description of a perfectly normal disc, with well-marked physiological cup.

We cannot indeed resist the conclusion, that this physiological cup (which, as is well known, varies greatly in size) has had to do duty for atrophy in not a few cases.

Space will not permit of a critical analysis of all the cases, but we may mention Nos. 25, (in which "atrophic changes were noticed," p. 228) 26, 27, 29, and 32, as instances in point: thus, of No. 29 it is stated: "Right eye: O.D., well defined by a narrow white rim of medium red tint, and having central bright white spot. Left eye: O.D., well-defined, very narrow white rim, it also has a small central white spot, the rest being of a medium pink glow."

As regards the main vessels, Mr. Aldridge leaves it to be inferred from his descriptions that in not a single case were these of normal calibre.

On the subject of General Paralysis, Dr. Gowers says,[1] "Most of the cases I have examined in various stages of the disease, presented perfectly normal conditions. In one case only was there the appearance of simple congestion of the disc."

Nettleship (quoted by Gowers) has recorded[2] an interesting case, in which optic nerve atrophy preceded the symptoms of General Paralysis.

[1] 'Medical Ophthalmoscopy,' 2nd edition, p. 178.
[2] 'Ophthalmic Hospital Reports,' vol. ix., p. 178.

A more recent observer, Dr. Lawford, has described (Transactions of Ophthalmological Society, vol. iii. p. 221), 7 cases of optic nerve atrophy in General Paralysis; it is not, however, stated from what proportion of cases these results were obtained, but only that 3 of the number were found in an examination of 22 cases of the disease.

It is interesting to note, in connection with the question of a relation between optic nerve atrophy and spinal symptoms in General Paralysis, that 5 out of these 7 cases had spinal symptoms, one posterior sclerotic, and 4 lateral sclerotic.

As regards insanity other than that associated with General Paralysis, the most important observations made in this country, so far as we are aware, are those by Dr. Clifford Allbutt, which are to be found in his work on the ophthalmoscope. Speaking broadly, we may say that Dr. Allbutt found a much larger proportion of changes than we have ourselves noted. Space will not permit of our analysing the cases in detail, nor indeed is it necessary to do this, as Mania is the only form of insanity with which an attempt has been made by the author to associate a definite change in the fundus oculi. We will briefly examine the main conclusions drawn in this case. Dr. Allbutt states, "that both in mania depending upon organic causes and in functional mania, the back of the eye if observed within a few days after a paroxysm, presents a vascular suffusion or pinkiness, so great after severe paroxysms as to obscure the discs." We regret that our attention was not forcibly directed to this statement at a sufficiently early period of our investigation to have examined it with the attention it deserves; so far, however, as our cases go they do not support the conclusion, for by a reference to what we have written under the head of Mania, it will be seen that we have examined three cases with reference to this point, in none of which was any abnormality detected. Dr. Allbutt further states "that during the paroxysm, on the contrary, the disc is anæmic, perhaps from spasm of the vessels;" this conclusion is, however, founded upon one observation only, and our own cases do not support it, for it will be remembered that we examined three cases during an acute maniacal paroxysm, in two of which the discs were perfectly normal, whilst in the third the changes

noted were in the direction of abnormal redness, rather than of abnormal pallor.

In Acute Dementia, Mr. Aldridge found "a state of retinal anæmia,"[1] and adds "the optic discs are pale." In most of our own cases the discs were normal; in one only did we note "tint inclining to pale, arteries rather small;" whilst in another case the discs were "very red." (See Table V., Nos. 9 and 10.)

In Epilepsy Mr. Aldridge states,[2] as the result of an examination of 102 cases, that "in a great majority of them a condition of hyperæmia of the retina and optic discs was found." As will have been seen, our own observations gave altogether different results.

Conclusions.—In all questions dealing with the condition of the fundus oculi in insane patients, it is necessary to draw a clear line of distinction between the disease known as General Paralysis of the Insane, and other forms of insanity. With this proviso we venture to draw the following conclusions:

1. That in Insanity proper (including all forms other than General Paralysis) changes in the fundus oculi are found in a small minority of cases; but that when allowance is made for changes depending upon associated constitutional conditions, errors of refraction, &c., the number of cases in which a connection between the mental (cerebral) state, and the accompanying change in the fundus oculi, can be so much as suspected, is very small.

As a corollary from this we may say,

2. That in Insanity proper no connection can be traced between the condition of the fundus oculi and the patient's mental state.

3. That in the majority of cases of "General Paralysis of the Insane," the fundus oculi presents a perfectly healthy appearance.

4. That in a minority of cases clear and precise lesions are found.

[1] 'Ophthalmoscopic Observations in Acute Dementia,' West Riding Asylum Reports, vol. iv. (1874), p. 296.

[2] 'The Ophthalmoscope in Mental and Cerebral Disease,' West Riding Asylum Reports, vol. i., p. 87.

5. That these lesions fall into two main classes, the one tending in the direction of slight neuritis, the other in that of atrophy.

6. That in the former class the affection declares itself as a hyperæmia of the discs, the edges being softened and indistinct, so that in some cases they can be traced with difficulty or not at all; and that these conditions tend—if the patient live long enough—to be replaced by atrophy, so that at length complete disorganisation of the nerve may take place. The changes are essentially chronic in their course.

7. That though atrophy of the optic nerves may thus succeed to a slight chronic interstitial neuritis, it is also not unfrequently primary *at the disc;* the atrophy may be complete, the patient becoming quite blind.

8. That the pathological basis underlying the appearances of slight neuritis, may be broadly characterised as a tendency to overgrowth in the connective elements of the nerve; the trabeculæ not only getting greatly hypertrophied, but the neuroglia corpuscles also becoming very large and numerous; these parts thus grow at the expense of the nervous elements, which subsequently atrophy.

9. That in the cases of primary atrophy, the pathological appearances eventually reached, though somewhat similar, may possibly take place in the reverse order *at the disc*; the nerve fibres being the first to dwindle, and the fibrous elements—trabeculæ &c., subsequently taking on increased growth.

10. That in a considerable proportion of the cases in which atrophy of the optic discs is met with, spinal symptoms are prominent in the disease, these symptoms pointing in the direction of posterior or lateral sclerosis of the cord; but that this connection is by no means invariable.

ON ALCOHOLIC PARALYSIS.

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THE effects of alcohol on the nervous system manifest themselves in various ways, chiefly according to the particular part of the nervous sphere attacked.

Whilst in many forms of chronic alcoholism the brain seems to be the chief organ to suffer, there is one affection, alcoholic paralysis or alcoholic paraplegia, in which cerebral symptoms are either altogether absent, or play a mere subordinate part, while most of the symptoms are referable to the spinal cord or to the peripheral nerves. The symptomatology of this affection has been well described in this country by Wilks ('Diseases of the Nervous System'), Lockhart Clarke ('Lancet,' 1872), Reginald Thompson, ('Med. Chir. Trans.' 1868), and more recently by Glynn, ('Liverpool Medico-Chir. Journal'), and Broadbent ('Lancet,' Feb. 16, 1884). On the Continent, the principal workers at this subject have been Leudet ('Arch. Gén. de Méd.' 1867), Lancereaux ('Gaz. Hebd.' 1865 and 1881), Moëli (Berl. Klin. Wochenschr. No. 14, 1884), and Charcot (see article by Féré in 'Prog. Méd.' June 14, 1884).

Our knowledge of the pathological anatomy of this affection is, however, still meagre, although the observations of Lancereaux, Moëli, and Broadbent, tend to show that the lesions concern chiefly, if not entirely, the peripheral nerves. As a small contribution to this interesting subject, I may be permitted to describe the lesions found in one case which was recently under my care, and to allude to a few cases which I have from time to time observed.

The disease runs by no means the same course in every case, nor does it invariably present the same grouping of symptoms, though certain of these symptoms are always, or almost always, present. Considering, however, the more prominent characteristics, we may, I think, divide alcoholic paralysis into certain types.

The first may be fitly designated *Alcoholic Ataxia*. Of this form I have seen several cases, and other observers have also alluded to it; thus Wilks ('Dis. of the Nervous System,' p. 225) mentions the case of a young woman "whose symptoms very much resembled those of locomotor ataxy, both in the character of the pains, which were like electric shocks, and also in the mode of progression." Leudet (loc. cit.) alludes to the difficulty of distinguishing such cases from true ataxia, and Lancereaux and Charcot also mention the ataxic symptoms. The cases of this description which I have seen were men, from 30 to 40 years old; the symptoms had come on more or less gradually, and consisted chiefly of marked inco-ordination in the lower extremities, absence of tendon-reflex, and lancinating pains. There was neither atrophy nor paralysis.

A patient whom I saw recently with Dr. Roe, of Patricroft, and who had been greatly addicted to alcohol for years, had already had one attack of paralysis, from which he completely recovered, after giving up alcohol. Having again fallen into bad habits, the old symptoms recurred (inco-ordination, lancinating pain, absence of tendon-reflex and slight loss of control over the bladder at night). Dr. Roe informs me that the patient's habits are still very intemperate, that his ataxic symptoms have remained unaltered, but that the patient is now suffering from rapidly advancing phthisis.

A case of very similar description I saw with my colleague, Mr. Jones: here also the symptoms came on after a series of alcoholic excesses, and I am informed have now quite subsided. The patient's family history was free from any neurotic taint, and he had not had syphilis at the time I saw him, though he has contracted it since then.

Another case I had an opportunity of watching for some time in the out-patients' department of the Manchester

Infirmary. A man, æt. 35, an excessive drinker, suffered from the symptoms described above, which came on in the course of three weeks, but subsided completely in three months. The recovery in his case was complete, the tendon-reflex being again established some months after all the other symptoms had disappeared.

These cases differ from those of the second type to be described below, in the absence of hyperæsthesia, hyperalgesia, vaso-motor disturbances, paralysis, atrophies and mental disturbances. The individuals attacked are chiefly men, and the symptoms disappear with the discontinuance of alcohol. In the diagnosis of such cases of pure alcoholic ataxia, one has of course to bear in mind how far alcohol may simply play the part of an etiological factor, and how far alcoholism and ataxia may be mere coincidences; moreover in the ordinary cases of ataxia, the symptoms, especially if they come on somewhat acutely, often show for a time a marked improvement. In the above cases, however, the ataxia followed so soon after the excesses, and so completely disappeared, that I have no hesitation in pronouncing them as alcoholic. This view is still further supported by the fact, that symptoms of ataxia are again seen in the second type to be described (especially the absence of tendon-reflex and the lancinating pains); moreover, some of the symptoms of ordinary locomotor ataxy, such as the arthropathies, the oculo-motor pareses, spinal myosis, &c., symptoms which we may consider spinal, are absent in the alcoholic ataxia. The symptoms observed harmonise well with the pathological anatomy which underlies the alcoholic paraplegia,—for as the changes observed chiefly concern peripheral nerves, we can understand the symptoms if we suppose that in the ataxic type we have an affection, a multiple neuritis, especially of the sensory nerves.

The second type of *alcoholic paralysis*, which deserves better the name of *paralysis*, is far more common, and is that which most observers have described.

This affection chiefly attacks females, who, though they deny the abuse of alcohol, have suffered from symptoms of chronic alcoholism (morning vomiting, chronic gastric catarrh, hæmatemesis, &c.), previous to the appearance of the nervous

troubles. These commence more or less acutely, and consist of sensory, motor, vaso-motor, and trophic disturbances.

The sensory disturbances are, as a rule, well marked and very characteristic, and consist of extreme hyperæsthesia of the feet and other parts of the lower extremities, with or without hyperalgesia; perversion of the sense for temperature (all objects when brought in contact with the skin feel cold); there are often present, in addition, lancinating pains in the lower extremities, and pain when pressure is applied along the vertebral column. The hyperæsthesia is often followed by anæsthesia or retarded sensibility, the hyperalgesia by analgesia.

The motor troubles consist of more or less marked paralysis, attacking the lower, and sometimes the upper extremities; in many cases the extensor muscles are chiefly affected, while in not a few cases the paralysis is more general. The superficial and mechanical reflexes are often diminished, the tendon-reflexes are nearly always absent; contractures have only been noticed by a few observers (Wilks, Buzzard). The vaso-motor affections consist chiefly of redness of the feet or hands, occasionally also of other parts of the body, and œdema, especially seen at the ankle, the dorsum of the foot, and the back of the hand. The nerves and muscles show marked degenerative reaction, though we have but few detailed statements on this subject.

Cerebral symptoms, such as insomnia, restlessness, more or less delirium and hebetude, though absent at first, often make their appearance during the course of the disease, and are frequently the cause of death, unless other complications should occur, such as phthisis, fatty degeneration of liver, &c.

Disorders of the circulatory, digestive and respiratory organs, are often seen during the progress of the disease, but are rather complications, or the primary effects of alcohol on these organs, than secondary manifestations of the nervous affection under consideration.

The prognosis varies very much. Many cases recover when they come early under the care of the physician, and when uncomplicated; some recover for a time, and afterwards relapse; whilst in others the disease pursues a steady down-

ward course, death ensuing from the cerebral symptoms, from exhaustion, or from some intercurrent affection, as is so often seen in other forms of chronic alcoholism.

The following case which was recently under my care, and which I was able to follow to its fatal issue, may be taken as a fairly typical example of this form of alcoholic paralysis, though it differs from some of the reported cases in the extent of the paralysis, which affected the lower extremities more uniformly than is ordinarily described.

The notes of the case were taken by one of my clinical clerks, Mr. E. E. Bowden.

Mary P., æt. 49, married, was admitted into the Infirmary on April 1, 1884.

Previous History.—The patient has resided in Manchester for 30 years; has had six children, all of whom have died, and has had one miscarriage. She has worked in a laundry, and admits having taken stimulants (though the patient denied any excesses, inquiries made of her neighbours satisfied us that she had been a chronic alcoholic; moreover, in her delirium she continually called out for beer). She has been in somewhat straitened circumstances, but enjoyed very good health up to ten years. She had small-pox some years ago, but has never had any other illness; the catamenia ceased two years ago, before which time they were very profuse.

History of Attack.—Two years ago the patient began to feel weak, especially in her knees, and she staggered then slightly in her walk; she also noticed that in dressing, when putting her garments over her head, she had to steady herself; she gradually grew weaker, and during the last few weeks before admission the weakness has so much increased, that she is now unable to walk.

State on Admission.—The patient lies on her back, and is unable to rise or turn over without great difficulty; her face has a haggard, almost demented expression; she is somewhat emaciated, and has a large bed-sore on her back.

The patient is fairly intelligent, and complains much of insomnia and slight headache; there are no other cerebral symptoms.

There is a considerable amount of paresis of the upper and

lower extremities, and also of the muscles of the back. The patient is scarcely able to walk or stand by herself, and with her eyes shut would fall immediately. She lies with her legs flexed, and extends them with great difficulty; can flex and extend her foot; can abduct and adduct the legs, but does so with difficulty and pain. The grasp of her hands is weak; can flex and extend her wrists and elbow; the effort of rising in bed causes great pain. There is great pain in both lower extremities, and patient shrieks when the legs are touched; the left knee is specially painful, the skin over it is reddened, and the joint is slightly swollen; there is also great pain in the back and loins, and along the whole of the vertebral column; there are no spots of either anæsthesia or analgesia; all the muscles seem equally atrophied and flabby; the superficial reflexes are fairly normal; the tendon-reflexes are absent; there is some diminution of the muscular sense in the upper and lower extremities.

The pupils are contracted, react very little to accommodation, and not at all to light. The tongue is furred and glazed; there is loss of appetite; the patient suffers from thirst and diarrhœa; the thoracic organs are found normal on physical examination; there is no cough and no expectoration; the liver and spleen appear normal on percussion; there is retention of urine, and the catheter has to be used; the urine is alkaline, has a sp. gr. of 1011, contains a large quantity of pus and a considerable amount of albumen.

The heart is found normal; the pulse regular, but quick, feeble, and easily compressible.

The temperature was in the morning 99, in the evening 100·4.

April 3rd.—The patient's condition is much the same as on admission.

Faradic Contractility.—Direct application to muscles; no contraction of the dorsal flexors of the right foot, even with the strongest current; those of the left foot react slightly to strong current.

Quadriceps femoris does not respond to any current.

Hamstring muscles and biceps react slightly.

Flexors and extensors of fingers react but slightly with the strongest current.

Biceps and triceps react somewhat better.
Erb's point scarcely any reaction.
Galvanic reactions.

LOWER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. brev. Dig. . . .	45	40	..
„ Exten. Prop. pol. } „ Tib. Antic. } . .	45	40	45
Right Tib. Antic. } „ Exten. Prop. pol. } . .	45	40	..
Left Rect. fem.	45	40	..
Right „ „	50	40	..
Left Hamstring	50	35	..

UPPER EXTREMITIES.

	Kat. Cl.	ACl.	A.O.
Left Ext. digit.	30	20	20
„ Triceps	20 (slight)	20	25
		25 produces tetanus	
Right Ext. dig.	30 (slight)	25	25
„ Triceps	35	30	..
Left Flex. digit.	35 (slight)	20	35
„ Erb's point	40	45	..
Right Erb's point . . .	40	..	45

Diagnosis.—Alcoholic paralysis. This diagnosis was arrived at without any great difficulty. The history of the case pointed strongly to a chronic alcoholic intoxication, and the symptoms on admission were well marked and typical. One might have thought of ordinary ataxia; but the extreme sensibility to pain and touch, and the degenerative reaction of the muscles, were against such an assumption. Against sub-acute or chronic poliomyelitis the sensory symptoms told with equal force. Against chronic meningo-myelitis pointed the fact that the disease commenced without pain, that paresis and inco-ordination were the first symptoms observed, and that the tendon-reflexes were absent. Against paraplegia dolorosa, such as is observed in cancer of the vertebræ, pointed the absence of any tumor, the absence of cachexia, the chronicity of the case, and the painless implication of the upper extremities.

There remained, therefore, scarcely any other disease but multiple peripheral neuritis, and with the history before us,

and the absence of any evidence showing that any other toxic agent, such as lead, arsenic, &c., was the cause of the symptoms (though such poisons would scarcely produce the sensory disturbances observed), we had no difficulty in arriving at the diagnosis stated.

Progress.—(I extract a few statements from short-hand notes.)

April 7.—The patient is much worse. She is delirious at night; her speech is slow and muttering; she lies with her legs flexed, and can only extend them with great difficulty.

April 9.—The patient is much worse; the delirium is now constant; the temperature 95: the extremities cold and cyanotic; pulse, very feeble, 80 per minute; the paralysis more extensive.

April 10.—Last night the patient became comatose, and died early this morning.

Autopsy, by Dr. Maguire, Pathologist to the Royal Infirmary. —Some fluid in left pleural cavity, a little fluid in pericardium; aortic valves showed some chronic thickening; the lungs, slightly emphysematous, contained a few caseous nodules in both apices. The liver enlarged, pale and soft. The kidneys showed several patches of caseation in the cortex; the pelvis full of pus; the capsule thickened, but peeled off easily, and showed the surface of the kidney affected by tubercular deposit. The bladder greatly inflamed, its inner surface covered with muco-pus, its walls thickened and infiltrated with tubercular deposit.

The brain was found normal, the pia slightly thickened; the dura mater showed numerous Pacchionian bodies.

The spinal cord and its membranes, when examined in the fresh condition, appeared perfectly normal.

Some peripheral nerves, which were reserved for microscopic examination, also appeared normal.

The muscles were pale, but otherwise normal.

After hardening the cord, I examined portions of it microscopically, without finding any changes: examined by Weigert's method ('Fortschritte der Med.' 1884), numerous small nerve fibres were found in Clarke's columns. The ganglion cells in all parts of the spinal cord were perfectly healthy.

Several posterior roots of the lumbar region were examined nd found perfectly healthy.

Of the peripheral nerves, there were examined the two sciatics, the musculo-spiral and the anterior crural. These nerves showed the characteristic changes described by Lancereaux in a very marked fashion. In the sciatic, longitudinal and transverse sections showed most markedly the breaking up of the myeline, and in many places the breaking up of the axis cylinder also. Longitudinal sections showed the segmental changes described by Gombault, and seen by me in a case of lead paralysis (see Dr. Moritz's paper in the 'Journal of Anat. and Phys.' 1881). The muscles showed no peculiar changes when microscopically examined. The post-mortem changes showed then a multiple peripheral neuritis, chronic tuberculosis of the lungs, and tuberculosis of the kidney and bladder of more recent date. The diagnosis of alcoholic paralysis was thus confirmed by the post-mortem appearances, which were exactly those described by Lancereaux and Moëli.

Though hyperæsthesia and hyperalgesia are prominent symptoms in this form of alcoholic paralysis, yet I think that both these symptoms may be occasionally absent, and the motor nerves may be principally affected. Thus, not long since, I observed the case of a man, æt. 38, who suffered from far-advanced alcoholic cirrhosis of the liver, and who in the course of a few days lost the use of his arms and legs. When admitted into the Infirmary, March 22nd, 1884, about three weeks after the onset of the paralysis, there was noticed, besides the ascites and enlarged spleen, marked atrophy and paralysis in the upper extremity, affecting chiefly the muscles supplied by the radial, the posterior interosseous and median nerves, and more pronounced on the left than on the right side. The back of the hand was œdematous. There was also observed some paralysis of the lower extremities, affecting chiefly the extensors of the toes and ankle. In the sensory system there was found a perversion of the sense for temperature, namely, the patient felt, as intensely cold, anything that touched his arms or his legs. (I find this symptom stated in several of the recorded cases of alcoholic paralysis.) There was absence of tendon and superficial reflexes.

The electric reactions of the muscles, carefully taken by Mr. A. Mumford, one of my clinical clerks, showed marked degenerative reaction. The special sense-organs were normal.

The cerebral symptoms consisted chiefly of sleeplessness.

The patient was in the hospital some time, without showing any improvement. He left, and died at home, and we had no opportunity of making an autopsy.

The nervous symptoms in this case resembled more those seen in chronic poliomyelitis; yet the history, the onset, the perversion of the sense for temperature, the marked degenerative electric reaction and the associated cerebral symptoms (though these might have been due to the cirrhosis), induced me to look upon this case as one of peripheral multiple neuritis, affecting chiefly the motor nerves and caused by alcohol. The combination of cirrhosis with alcoholic paralysis has already been noticed by Lancereaux ('Gaz. Hebd.' 1865).

The *Anatomical lesions* then which underlie alcoholic paralysis, to judge from the post-mortem results of Lancereaux and Moëli, and those given in the case above, consist of a peripheral multiple progressive neuritis. Broadbent, in his case, found the cord normal, but was not allowed to examine any other part of the body. In one of Lancereaux's older observations the antero-lateral columns of the cord were found slightly sclerosed; in all the later observations the cord was found healthy.

The post-mortem lesions explain the symptoms very well, and also account for the diversity which we observe, according as the one or the other set of nerves are chiefly involved.

As regards *differential diagnosis*, the disease resembles in many respects the progressive multiple neuritis described by Leyden, Strümpell, Pierson and others, of which indeed it forms simply a particular species. It differs, however, from this non-alcoholic multiple neuritis in several particulars; it runs a less acute course, is less progressive, the paralysis is more confined to the extensor groups of muscles, and though there is a considerable amount of pain in the non-alcoholic form, there is not usually the very characteristic hyperæsthesia and hyperalgesia described above; the cerebral symptoms also are absent in the non-alcoholic form.

It further resembles other intoxications, notably those produced by lead.[1] Lancereaux has long ago drawn attention to this, and, pathologically considered, the lesions are the same in both; just as in alcoholic paralysis, so in lead paralysis, we have to do chiefly with peripheral nerve changes; though lead seems to select the motor nerves *par excellence*. The similarity of the action of lead and alcohol is further evident when we consider other diseases in which both play the important etiological element, as in gout, in the true cirrhotic kidney, and, I may perhaps also add, chronic endocarditis and some brain affections.

The *treatment* of alcoholic paralysis, which is often successful in the less advanced and uncomplicated cases, must have for its prime object the withdrawal of the alcoholic stimulant. Of other remedies, the opiates, cinchona (recommended by Leudet and Wilks), strychnia, hydropathy and the application of electricity seem indicated.

The iodide of potassium, which I have tried in some cases, has not given me any very encouraging results.

I wish now to draw attention to certain *visceral neuralgias* which, either alone or in combination with other nervous symptoms, are found in chronic alcoholism, and which I believe are caused by the alcohol. Thus, I was asked to see a lady who was an alcoholic, and who suffered from very intense colicky pains; these attacks would last for days; they were unaccompanied by vomiting or any other gastric disturbance. There was marked constipation. There was no reason to suppose lead-poisoning, nor did the gums show any blue line—yet iodide of potassium was freely given without benefit. Opium gave some temporary relief; the pains, however, recurred from time to time, and only disappeared when the patient was put under restraint, in her own home.

Another lady, aged 27, who was suspected to be an alcoholic, suffered from attacks which resembled very much angina pectoris; there was excessive cardiac dyspnœa; the pain radiated into the left arm, slightly also into the right, and there was also some epigastric pain. Physical examination of

[1] Atrophies produced by arsenic have been noticed by Gerhardt, 'Cbl. f. Klin. Med.' 1882.

the chest-organs showed nothing abnormal, and beyond a very visibly pulsating abdominal aorta, nothing abnormal could be detected in the abdominal cavity. The cause of these symptoms was very doubtful to me; but when they disappeared, along with some other neuralgic troubles (chiefly affecting the trigeminus) after the patient was put under restraint in a home for dipsomaniacs, I strongly suspected that they had been due to alcohol; in this view I was confirmed, for the symptoms reappeared, when the patient left the home and recommenced her old habits. She was placed a second time into the home, and again improved; but died subsequently from pneumonia. I had no opportunity of making a post-mortem examination.

These visceral neuralgias again resemble those produced by lead. Lead colic is common enough, and in a small epidemic of acute lead-poisoning produced by the inhalation of particles of lead-chromate, I have seen two cases, where, after the acute symptoms had passed off, symptoms very much like angina pectoris came on. These symptoms resemble most a vaso-motor disturbance, and as such we can easily understand their appearance in chronic alcoholism.

ON ATROPHY OF THE BRAIN IN IMBECILES.

BY FLETCHER BEACH, M.B., M.R.C.P.,

Medical Superintendent of the Dareuth Asylum.

MANY writers speak of two forms of atrophy of the brain, and it is a convenient method of describing the disease. In the first, there is incomplete development; in the second, there is loss of nervous elements which had previously been present. Taking first that variety in which there is incomplete development, and excluding that form of it in which the hemispheres are completely wanting and vegetative life merely exists, I would remark that cases illustrating this condition are commonly found in Asylums for Imbeciles. Microcephalic imbeciles are instances of this class. Microcephaly may be general or partial. Some portions of the brain may be too small or altogether absent. I have found the convolutions of certain parts very much reduced in size from non-development, while the remaining portions are too large or normal. The occipital lobes are usually arrested in growth in microcephalic cases, and the island of Reil may be left uncovered. The corpus callosum is sometimes shortened posteriorly, and cases are on record where this portion of the brain has been wanting and the commissures deficient.

As an instance of the arrest of growth of the occipital lobes, I would refer to a case related by Dr. Shuttleworth in the 'Journal of Mental Science for 1878,' in which the frontal and parietal lobes were comparatively well-developed, but the occipital lobes were quite rudimentary.

Usually, however, microcephaly is general, and the deficiency consists in the smallness of the hemispheres. In the 'Transactions of the International Medical Congress' for 1881 I have related two cases, in one of which the brain when removed from

the body weighed only 7 ounces, yet a minute examination of it showed that nearly all the convolutions were present, though very small in size. In the other, the brain weighed 20½ ounces. Two brains of microcephalic cases have since come under my notice, and these weighed 20¼ and 24½ ounces respectively. The nerves of special sense are usually well-developed in microcephaly, and the ganglia of the base and the spinal cord are of nearly normal size. The cerebellum is relatively much larger than in the normal brain, often being in the proportion of 1 to 3 or 4, the ordinary relation being 1 to 8.

The following are illustrative cases:

G. L., aged 13 years, admitted into Darenth Asylum, January 19th, 1878. The father is a very hard drinker and often ill-uses the mother. He has an impediment in his speech, and so have all his side of the family. The mother is a temperate woman, but has a brother in Colney Hatch Asylum. Intemperance and hereditary predisposition to insanity are therefore present. The parents are not connected by consanguinity. G. L. had a small head when born. He did not walk till 5 years old, and has never spoken. The mother ascribes his condition to ill-usage by the father during pregnancy. He is a twin; the other child is dead, but his head was of normal size. There have been twelve children, of whom one, a girl aged 8 years, is also microcephalic. Seven have died, and of these one was microcephalic and died in convulsions.

On admission, G. L. was found to be rather thin, with a muddy complexion and a bird-like aspect. His head measured 17⅜ inches in circumference (3 to 4 inches below the normal size), 10⅞ inches transversely (3⅞ inches with calipers), and 11 inches antero-posteriorly (6 inches with calipers). Since his residence in the Asylum his head has increased a little in size, being now 18⅝ inches in circumference, 11 inches in the transverse diameter, and 11⅛ inches in the longitudinal direction; but even now the outline of it, when compared with one of normal size, shows the great difference at once. His forehead was 3⅝ inches in breadth. He had good use of his limbs and was not subject to fits. In temper he was some-

what stubborn, but was easily managed. He could not speak, but could only make a sound like "ah." He was able to take his food without assistance, and could help in the ward, but he could not wash or dress himself without assistance. He had some power of observation, imitation, and attention, though but little memory. He went to school in the Asylum daily, and at the end of one year had learnt some letters of the manual alphabet, and could hold up one or two fingers to correspond with the objects shown to him. Two years afterwards he was able to read some words with the manual alphabet, could spell "cap," write "a, o, d" from memory, hold up fingers as far as four to correspond with objects, and could match colours and forms. He had also learnt to make himself very useful in household work, and could sew on buttons, and do simple repairs of clothing. Considering the size of his brain, I think we may say he has made fair improvement, though not so much as his sister, who has a smaller head.

A. P., admitted August 15th, 1877, was three years old on admission. Her parents are healthy, temperate, and not connected by consanguinity. A brother of the mother is subject to fits. The mother attributes the patient's condition to a fright which she had when four or five months advanced in pregnancy. This is the fifth child. The others are all healthy and have no mental defect. The child's head was small when born, and the mother thinks it has not increased much in size since, though the body has grown normally. She has never noticed passing objects, and has never been able to stand or hold anything. She is not subject to fits. Her thumbs have been drawn into the palms of her hands since birth.

A. P. when admitted was a fairly nourished child, of fair complexion, 30 inches in height. Her head measured $15\frac{1}{4}$ in. in circumference (5 to 6 inches below the normal size), $9\frac{1}{2}$ in. transversely, and $10\frac{1}{2}$ in. antero-posteriorly. As in the preceding case, the head has grown in size since admission, the corresponding measurements to those above now being 16, $9\frac{5}{8}$ and $10\frac{5}{8}$ inches. Her forehead is small and receding, tending to the oval in shape. In height it is 2 inches, and in width 3 inches. Her arms and legs are of normal size, the

former measuring 11 inches, and the latter 13 inches in length; she cannot stand, though she can kick her legs about. She is subject to spasmodic retraction of the limbs, and though she can move her arms, she cannot hold anything. Her forearms are contracted on the arms and the fingers are clenched. Her legs are also contracted on her thighs, and cannot be straightened. She is placid in disposition and cheerful. Her habits are faulty. Her mental capacity is of course very small, but there is some power of observation and a little of attention. She has now been 4½ years in the Asylum, and during the whole of the time has been kept in the Infirmary, where she sits by day coiled up in a chair, and requiring to be fed, clothed, and generally attended to. She now seems to recognise her nurse, and laughs when pleased, but the improvement made is not great.

Both of these cases, and especially the latter, have heads much below the normal size, and correspondingly the brains are deficient in development. It is not, however, only the smallness of the quantity of the brain, but deficiency in quality which is involved. If we look at sections exhibited under the microscope, we shall see the great difference between a normal and a microcephalic brain. In the latter we notice the deficiency of cell processes, and the retraction of the protoplasm in the cell, accounting for the spaces so often visible. As to the causation of microcephalic imbecility, it has been held that it is due to the sutures of the skull closing in prematurely, and so preventing the growth of the brain; but, in opposition to this theory, there is the fact that many cases have been collected with open sutures. "Even in those cases where the sutures have closed in before birth, the question still remains whether the brain ceased to grow because the sutures are closed, or whether the sutures closed in because the brain ceased to grow; or lastly, whether both the brain and its coverings ceased to grow from a common cause."[1]

The second form of atrophy, viz., that in which there is loss of nervous elements which had previously existed, may present itself in various forms, but the most interesting is perhaps that in which there is atrophy of one side of the brain, usually the

[1] Ireland on Idiocy and Imbecility.

left, with coexistent atrophy of the limbs of the opposite side of the body. Now, though imbecility is not necessarily the result (vide cases quoted by Van der Kolk), yet examples of this disease are of fairly frequent occurrence in Asylums for Imbeciles. According to this author, everything depends upon the more or less healthy state of one hemisphere of the brain. "If, as from the nature of the case seldom occurs, the inflammation and affection of the pia mater has not extended to this hemisphere, if the grey matter under the cerebral convolutions has here continued perfectly sound, there is no reason why this remaining hemisphere should not be able to act without impediment in the exercise of those functions which are necessary to our mental powers, just as one eye sees as sharply though the other be lost. But where grey matter is injured in both hemispheres, particularly anteriorly, disturbance of the intellectual faculties will be inevitable."

An inflammation of the brain, meninges, or skull during fœtal life or early childhood, no doubt will cause the disease. The paralytic form of imbecility, for instance, seems to depend upon an atrophy of the brain caused by chronic meningitis or inflammatory processes in the cortical substance. Either of these causes may of course occur before birth or come on afterwards.

In the following case I believe atrophy of the brain to exist, and I think there is no doubt that it was produced before birth.

W. B., aged 9 years, was admitted into Darenth Asylum, November 14th, 1877. The parents were temperate and healthy people, and not connected by consanguinity. When the child was born, his right side was found to be powerless, in consequence, as is supposed, of the mother during pregnancy falling down and striking her side with great violence against a wall. On admission, his head was found to be flattened on the left side, but of fair size, being $19\frac{7}{8}$ inches in circumference. He limped with his right foot in walking, and the fingers of his right hand were flexed in the palm. His mental capacity was fair. He could read a few letters, print with his left hand "a" and "o," count to 20, and knew a few colours. He has made

good progress in school, so much so, that he is now able to read well from the 1st Standard, write and transcribe (with his left hand) in a book, work easy addition, subtraction and multiplication sums, and recognise all the colours and shades, and easy and difficult forms. He has progressed equally well in the tailor's shop, and, as a result of his industrial work, he is now able to straighten the fingers of his right hand, and make use of this hand in dressing himself and in his work.

The progress in such cases is nearly always fair mentally, though but little physically. In the one just quoted, greater physical improvement than usual has been made, but there are even now marked differences in length and size between the limbs of the healthy and those of the affected side.

RIGHT ARM (AFFECTED SIDE).

	Inches.
From point of acromion process to external condyle of humerus	10½
From external condyle of humerus to styloid process of radius	8½
From centre of wrist to end of middle finger	6⅞
Circumference round centre of deltoid	8
„ „ „ „ „ biceps	7½
„ „ „ forearm 1½ in. in front of inner bend of elbow	7½
„ „ „ forearm 1 in. in front of wrist	5⅝

LEFT ARM (UNAFFECTED SIDE).

From point of acromion process to external condyle of humerus	11¼
From external condyle of humerus to styloid process of radius	8½
From centre of wrist to end of middle finger	7½
Circumference round centre of deltoid	9½
„ „ „ „ „ biceps	9
„ „ „ forearm 1½ in. in front of inner bend of elbow	8¾
„ „ „ forearm 1 in. in front of wrist	6½

Every measurement of the affected arm is seen to be less than that of the unaffected one, and so with the leg.

RIGHT LEG (AFFECTED SIDE).

	Inches.
From inferior spinous process to external condyle of femur	15
From external condyle of femur to external malleolus	14
Length of sole of foot	9½
Circumference of thigh 10 in. below inferior spinous process	14½
„ „ „ calf 5 in. below external condyle	10½

LEFT LEG (UNAFFECTED SIDE).

	Inches.
From inferior spinous process to external condyle of femur	16¼
From external condyle of femur to external malleolus	14½
Length of sole of foot	10
Circumference of thigh 10 in. below inferior spinous process	15⅝
„ „ „ calf 5 in. below external condyle	11¼

In the next case the date of the onset of the paralysis is doubtful. E. H., aged 18 years, was admitted into the Clapton Asylum, May 3rd, 1875, having been transferred from the Hampstead Asylum. I am indebted to Dr. Orange, Medical Superintendent of the Broadmoor Criminal Lunatic Asylum, for the following history. The father of E. H. had died in 1868, of cancer of the bladder. The mother was a patient in the Broadmoor Asylum, having killed one of her children while insane. The parents had been temperate people and were not connected by consanguinity. There was no family history of epilepsy or paralysis. This was the eldest child. There had been one other, who was killed by the mother, as mentioned above. E. H. was of sound mind at birth, and was doing well at Southall School, but was sent to Hampstead Asylum in consequence of epileptic fits, from which she suffered. These commenced at the age of 2½ years (cause unknown). Her intellect had become affected by the fits.

On admission, she was a fairly nourished girl of dark complexion, with loss of power of the right upper extremity (the forearm being contracted on the arm, the wrist on the forearm, and the fingers flexed in the palm of the hand), and weakness of the lower one on the same side. There was no means of ascertaining the cause of the paralysis, or when she was first afflicted with it. She was still having epileptic fits. She talked and answered questions with considerable intelligence, was of a quiet disposition, had pleasing manners and was a general favourite. Her mental capacity was very fair for an imbecile. She could read from the 3rd Standard, and write very well with her left hand. She had made considerable advance in arithmetic, being able to do simple and compound reduction sums. She was fond of music, and knew all the colours. From this it will be seen that she was an imbecile

of a very high type. She made progress at school in the intervals between the fits, and was able to sing at the entertainments. In October of the same year she was noticed to be getting very thin, and on examination the physical signs of phthisis were discovered. She now had attacks of petit-mal very frequently and became very emaciated. She died on the 21st of December, 1875, having been unconscious the preceding day for 6 hours. The post-mortem examination was made 12 hours after death. The calvaria was removed, and the convex surface of the brain examined in situ. On stripping off the dura mater, the left hemisphere was seen to be much wasted, and the arachnoid membrane over its surface was in places thickened and opaque. The wasting was chiefly noticeable in the frontal and parietal regions. The texture of the hemisphere was evidently altered, for, to the touch, the left was hard and firm, while the right was elastic and apparently normal. The left middle lobe was much reduced in size, measuring only 1 inch transversely, the right measuring $2\frac{1}{4}$ inches in the same direction. There did not appear to be excess of fluid in the left subarachnoid space, but as some drained away while removing the calvaria, the defect may be thus accounted for. The brain was then removed and found to weigh $28\frac{3}{4}$ ounces. A quantity of fluid drained away during the process of extraction. A depression as large as a small pear could now be seen on the convex surface of the posterior half of the left hemisphere. On slicing through the brain transversely, the left lateral ventricle was found to be enormously dilated, measuring $4\frac{1}{4}$ inches in length and 2 inches in breadth at its posterior (most dilated) part, the right ventricle measuring only $3\frac{3}{4}$ inches, and 1 inch in the corresponding directions. The depression above mentioned was now seen to be due to the roof of the left ventricle falling inwards. Its upper wall in this situation was not more than $\frac{1}{4}$ inch in thickness, the roof of the right measuring $\frac{3}{4}$ inch. In consequence of the dilatation of the left ventricle, the left choroid plexus in its course outwards dipped downwards one inch anterior in position to the right. Comparing the two hemispheres, it was found that the right measured 7 inches in length, and $2\frac{1}{2}$ inches in breadth, but the left only $6\frac{1}{4}$

inches longitudinally and 2¼ inches transversely. The right hemisphere weighed 15¼ ounces, the left only 5¾ ounces. The convolutions on the right side were exceedingly coarse, while those on the left side were smaller than usual for a child of her age. The white matter of the anterior and middle lobes on this side was reduced to a mere line. Unfortunately I did not examine the corpus striatum and optic thalamus of the two sides.

Examining now the cranium, the left side was found to be much thicker than the right, and the internal surface presented marked differences on the two sides. Thus the left anterior fossa was more prominent than the right, the left anterior lobe of the brain being correspondingly depressed inferiorly. The left middle fossa was much smaller than that on the opposite side and was pear-shaped, while the right was more quadrangular. On measuring the two, the left was found to be 1½ inch in length and 1¼ inch in breadth, the right measuring 2 inches and 1¾ inch in the same directions. The left posterior fossa was shallow. The left cerebella fossa was larger than the right. The lungs presented cavities in the apices, and patches of tubercle and caseous material were scattered through the substance of both.

The cerebellum was not specially examined, but from the fact that the left cerebellar fossa was larger than the right, there is no doubt that the left lobe of the cerebellum was the larger of the two.

The third case, A. H., æt. 16 years, was admitted into the Clapton Asylum, May 5th, 1875, and died November 12th, 1875. Her father had died at the age of 33, of typhoid fever; the mother was alive and healthy. There was no family history of neuroses, but the aunt on the mother's side had died of phthisis. Both parents had been temperate, and were not connected by consanguinity. There were three other children who were physically weak, but who were unaffected mentally. The patient is said to have been mentally sound when born, and nothing was specially noticed (except some spots on the body, when the child was a month old, due to the father having had syphilis before her birth) until four years of age when the child had a heavy fall, and the right side of her

body became paralysed. Her intellect, however, was not noticeably affected until the age of 12 years, when she became subject to fits (cause unknown). These have continued ever since, but are slight in character. Since the onset of the fits she has become dull. On admission she was found to be emaciated and the right side of the body was paralysed. She could speak indistinctly, was quiet in disposition, but dirty in her habits. Her mental capacity was small. She could only read words of two letters, spell A B C, add to 6, count to 40, and recognise two colours. She was unable to dress, or take her meals without assistance. She went to school daily, but made little progress. She was very feeble, and frequently had attacks of diarrhœa and tonic contractions of the right side, no doubt epileptic in character. Later on, twitchings of the lip were noticed, and the left side of the body sometimes became affected. She died exhausted, November 12th, 1875. At the autopsy, the body was found emaciated and the right arm flexed on the forearm. The cranium was much thicker than normal. (I find I have not noticed whether it was thicker on one side than the other.) The brain weighed 29½ ounces, and was smaller than normal for a child of her age. The convolutions were flattened, and the sulci diminished in depth. There was a large excess of subarachnoid fluid, especially over the left hemisphere of the brain. The arachnoid membrane was opaque in places, especially over the site of the subjacent vessels. On slicing through the brain, the lateral ventricles and the fourth were seen to be much dilated with fluid, though some had drained away, the left lateral ventricle being apparently the fuller of the two, so that the septum lucidum was a little to the right of the middle line. On comparing the two sides of the brain, the left hemisphere appeared smaller than the right, especially anteriorly. The corpora striata, optic thalami and cerebellum appeared normal and equal in size, but this may have been due to the atrophy of the hemisphere being small in amount.

From the cases just given, as well as those related by Van der Kolk, Dr. Taylor in Guy's Hospital Reports, and others, it appears that the usual appearances found post mortem are: thickness of the cranium, opacity and thickness of the mem-

branes, effusion of serum into the subarachnoid space, sometimes into the ventricles and atrophy of one hemisphere, including the corpus striatum, optic thalamus and pons of the affected side. Since the fibres of the superior peduncles of the cerebellum undergo a complete decussation beneath the upper pair of the corpora quadrigemina, and those of the middle peduncles decussate in the pons varolii, while the fibres of the pyramids of the medulla have their well-known crossed direction, there is atrophy of the cerebellum and of the spinal cord on the opposite side.

The course of events appears to be this; first, there is, as the result of chronic inflammation of the meninges, or of the cortical substance, wasting of one side of the brain. To compensate for this the skull becomes thickened, and serum is poured out beneath the arachnoid and into the ventricles. Then, since those parts of the brain which are connected with motion are wasted, the limbs whose action is governed by them are imperfectly nourished and become atrophied.

With reference to the microscopical appearances found in atrophy of the brain, a case is quoted by Dr. Major in the 'Journal of Mental Science' for 1879, in which especial attention was paid to this point, with the following result.

The cortex of the convolutions of the affected side was seen to be reduced in thickness, and under a low power seemed to be made up of small round cells of uniform size, here and there only a pyramidal corpuscle being observable, in marked contrast with the appearances in the cortex of the opposite hemisphere. Examination with higher powers showed that the nerve-cell elements were extremely few, as well as small, ill-developed, and deficient in branches. The intercellular matrix of the neuroglia was denser and coarser than in the right hemisphere and under normal conditions. In the internal white matter the nerve fibres were nearly absent in some places, the whole tissue being represented by numerous Deiter's connective tissue cells with their dense intercommunicating net-work, naked nuclei and occasional vessels. The atrophied lobe of the cerebellum showed a condition of thickness and wasting of the outermost grey layer, few and ill-developed cells of Purkinje, and excess of connective tissue corpuscles.

The spinal cord was not examined by Dr. Major, but was so by Dr. Taylor in a case related by him. There was shrinking of the anterior grey cornu and of the antero-lateral column of the affected side, but the number and size of the ganglion cells of the anterior cornua were almost identical. The cells on both sides were identical in colour after staining, in structure, outline, and processes, with those of the opposite healthy side. There was nowhere any degeneration of the nerve fibres in the white columns, and the dorsal and lumbar regions were distinguishable in no respect from those of a healthy cord.

ON THE ALTERATIONS OF CORTICAL EXCITABILITY BY COLD APPLIED TO THE SURFACE OF THE BRAIN.

BY H. DE VARIGNY, M.D. (PARIS.)

ALTHOUGH several physiologists have already investigated this question, there does not seem to be any general understanding as to the interpretation of the results obtained. I have recently made some ten or twelve experiments concerning this *quæstionem vexatam*, and I made them—purposely—before acquainting myself with the conclusions reached by others, but knowing the methods on which they proceeded in their experiments.

M. Marcacci, an Italian physiologist, has investigated the subject, if not thoroughly, at least more so than any one else. His experiments are described, by himself, in the 'Archives Italiennes de Biologie.'[1] They were intended to be used as facts in support of, or—as the case might be—against, the theory of experimental excitability of the gray matter of the brain. The nervous tissue, and especially the gray matter, must be considered as the most exalted of all the tissues, the last to be evolved, the one the functions of which are the most perfect and complete. It must also be the most delicate, that is, the one injury to which is of most consequence to the organism, as well as the one most easily injured. It is known in a general manner that all tissues die rapidly when submitted to the influence of a certain degree of cold, or of heat: M. Marcacci believes that the same is true of the gray matter; and we all agree in this, although not being certain which degree of cold—for instance—is enough to deprive the gray matter of its vitality.

But we do not know positively whether cold applied to the surface of the brain in the way in which M. Marcacci applies

[1] Vol. i., p. 261, 1882. "Etudes Critiques et Expérimentales sur les Centres moteurs corticaux." See abstract in 'BRAIN,' 1883, p. 112.

it does really kill the brain-elements: in fact, I do not believe it does; nor do I believe I killed them in my own experiments conducted in the same manner as M. Marcacci did. This author says—and it is true—that by pulverising ether, or other liquids, such as chloride of methyle, on the brain surface, one may obtain 20, 30, even 40 degrees below zero (Centigrade).

It is true that if such liquids are pulverised upon a thermometer, the cold may be intense enough to bring the mercury down to $-30°$ or $-40°$, but are we sure that the same process can freeze the brain surface? I have often pulverised ether on this surface during ten and twelve minutes continuously; I never found the gray matter frozen; the blood did not cease to flow in the vessels, and the temperature always came back to its normal degree in a few minutes.

The surface was certainly cold after pulverisation, but it never remained so. I think it must be a very difficult process to really *freeze* the cerebral matter, even to half or quarter of an inch in depth, on the living animal. After my own experiments I feel quite sure that I never froze the brain-surface, nor even was near doing so, and I do not think the case to have been different with M. Marcacci. He shows that the pulverisation of ether, etc., on the brain-surface does not interfere with the excitability of the same; it even seems in some cases that excitability is increased. M. Marcacci concludes that when the brain-surface is excited (with electricity), the motor results are to be ascribed not to the excitation of gray matter, since its movements are obtained even where this matter is [thought to be] frozen, but to the excitability of the subjacent white tracts. I have already discussed the results obtained by M. Marcacci, in my work on the electrical excitability of the brain:[1] I shall not go over them again here. But I must remark that this observer's conclusions do not logically follow from his experiments. He shows that motor results are still obtained when the gray matter has been submitted to the influence of cold: we admit this; it shows that if the gray matter is paralysed, the white

[1] 'Recherches expérimentales sur l'excitabilité électrique des Circonvolutions cérébrales, et sur la période d'excitation latente du cerveau.' Paris, Alcan, 1884.

one is not; but does it prove anything more? Does it show that the gray matter is necessarily and always unexcitable?

In my own experiments, I have worked on dogs, and used the ether-spray. The way I proceeded was the following. The dog being narcotised with chloral (intra-venous injections), and the brain being exposed, I ascertained exactly what strength of current was necessary to obtain a motor-reaction of the fore-leg, for instance, and also the time required for the occurrence of this reaction—that is, the time elapsing between the moment when the brain was excited and the moment the motor result began: in short, the latent time of reaction. When these two points had been accurately ascertained, I began pulverising ether on the brain-surface, doing all I could to refrigerate the motor-centre of one leg only, for instance, without injuring that of the other. When refrigeration had been going on during five, ten, or fifteen minutes, I ascertained whether the motor-centre could be excited, with what current strength, and with what latent time.

I shall quote here some of my experiments.

Experiment 1.—Spaniel dog; weight, about six kilogr. Young, not very lively. The motor-centre of the fore leg is easily excited by Dubois-Reymond's secondary coil at 5° Cent. The latent time is 0·07 second.

I pulverise ether during eight minutes. I then try to excite the motor-centre with the same current (5° Cent.), no result. I then push the secondary coil gradually from 5° to 0°. Unfortunately the experiment had to stop here: I did not ascertain how much time was required for the recovery of the excitability of the motor-centre.

Experiment 2.—In this experiment I obtained the same results as in the preceding; that is, the motor-centre lost its excitability after a few minutes, and could no more be excited by the current which was previously sufficient, nor by the strongest current that could be obtained with the coil. However, after a little while, some reaction could be obtained.

Experiment 3.—In this experiment I obtained a very interesting result. After having ascertained the two data necessary in this sort of experiments, I sprayed ether during eight

minutes. During five minutes after this, the motor-centre was entirely unexcitable. But within a short time it seemed to react again, and about a quarter of an hour after the spray the current required was the same as before. As soon as I perceived that excitability had returned, I ascertained the length of the latent time, and to my great astonishment found it shorter than I had ever done before the pulverisation. Instead of 0·09 second and 0·08 second, I found 0·07 second and 0·06 second. It seems as if the temporary paralysis of the nervous elements had been followed by a kind of increase of energy or hyperæsthesia.

I need not relate any more experiments here, but may confine myself to giving the general results reached by me. Generally speaking, after a five to ten minutes' pulverisation, excitability disappears or only diminishes. It can always be made to disappear if the spray be kept on long enough. After some while, if the cold has not been too intense, it reappears slowly, and may become greater than it was before. At any rate I have never seen a five to ten minutes' pulverisation injure the *cortex cerebri* to such an extent that recovery of excitability—transitory perhaps—became impossible. M. Marcacci either must have used very strong currents, capable of exciting the white tracts when the gray matter was paralysed, or—as I should feel inclined to believe—did not really paralyse the gray matter. I do not see how to explain otherwise the interesting results this physiologist has obtained. I cannot say anything definite as to the length of the latent time after the return of excitability; I have generally found it about the same, sometimes a little longer, and once much shorter.

Clinical Cases.

CASE OF OCCIPITAL ENCEPHALOCELE IN WHICH A CORRECT DIAGNOSIS WAS OBTAINED BY MEANS OF THE INDUCED CURRENT.

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My excuse for publishing a case of Encephalocele, a condition the nature of which is well understood, lies in the fact that in this particular instance, as in all hitherto published, the *details* of diagnosis were very obscure, and I therefore ventured to clear up the difficulty by stimulating the hernial portion of brain with the interrupted induced current. Wishing also to draw attention to the causes of failure in those cases where operation was attempted, it seemed best to take this opportunity of discussing the various methods and their results.

I am greatly indebted to the kindness of Mr. Marcus Beck for the opportunity of treating this case.

CLINICAL REPORT, SEPT. 22ND, 1884.

ERNEST JOHN O., No. 1921.—Admitted into University College Hospital on 21st September, 1883. Age six weeks.

Past History.—The child was born at full time, the labour was easy, and nothing abnormal noticed, excepting the tumour which was at the time of birth equal in size to half the child's head. Child was "jaundiced" three days.

Family History.—Patient was the fourth child in five years, the last child being 16 months old. The second child died, cause not stated; the remainder were healthy. The father (an ostler) was a well-developed, healthy young man. The mother was of average height, but thin and poorly developed. Patient's maternal grandmother and three aunts died of "consumption." Maternal uncle now in Brompton Hospital with phthisis Father's family healthy.

Present State, on admission.—Patient was a pale thin child,

with a pendulous tumour attached to the occiput. All its life-processes appeared perfectly normal, except that its temperature (rectum) varied from 97° F. to 99·4° F. as a "normal" range.

Head.—The skull was very prognathous and scaphocephalic. the masto-temporal ridge and lambdoid suture very strongly marked, while the fontanelles were perfectly normal. The scalp was covered with soft hair, and hanging from the occipital region was a sacculate tumour, which covered the lower two-thirds of the occipital bone, and occupied about half the breadth of the bone in the middle line. The tumour was covered by skin, the surface of which was harsh in patches, and reddened all over, with a few enlarged veins showing, close to the attached border. It was also hairy (like the rest of the scalp) for about one-fourth of its surface, extending from the attached border. Moreover, on the right side of the sac there was a large firm cicatrix (congenital) which was 3 in. long by ¼ in. broad, situated opposite the middle, and directed backwards and slightly upwards. The circumference of the sac at its greatest diameter was about 12½ in. The wall of the sac was composed of skin, and a strong, evidently fibrous capsule with trabeculæ crossing the cavity at different points. The occipital bone could be easily felt to be perforated by a hole apparently 1 inch in diameter, and occupying one-third of the occipital crest. Emerging from this hole, and projecting into the fluid which filled the tumour, could be felt a rounded mass, apparently 1¼ inch broad, but (owing to the fluid around it) its outline could not well be made out. The rest of the surface of the body, the skeleton, the muscles, and viscera, appeared perfectly normal.

Central Nervous System.

Cerebrum Cortex.—Child appeared to possess the average mental capabilities of its age. It slept soundly, but when awake there was ptosis on both sides, so that the eyes were only half open. It took little notice of things around, and rarely made any purposive movements. When the chest was tickled, it (basal ganglia?) made semi-coordinated movements (defensive) of all the limbs. When awoke from sleep, its limbs performed irregular movements of flexion and extension, and sometimes the whole trunk also.

There was constant rigidity, with contraction of the flexor muscles of the left forearm, and slightly of the arm also. The elbow was kept flexed at a right angle.

Medulla Oblongata. Respiratory Centre.—Respirations normal. Firm pressure on the sac caused arrest of respiratory movements in full inspiration.

Cardiac Centre.—Heart beat perfectly regular. Only slight quickening of the pulse could be made out on pressure of the sac.

Vaso-motor Centre.—Skin over trunk and limbs moist and warm. Tache cérébrale well marked over the whole body.

Vomiting Centre.—Patient never vomited.

Deglutition Centre.—Swallowing perfect.

Cranial Nerves.

1st. Patient apparently noticed odours such as peppermint, &c. (Substances employed were not so strong as to irritate the 5th nerve.)

2nd. Patient took no notice of bright objects held or moved about in front of the eyes, and the pupil was not noticed to vary under those circumstances.

3rd. The pupils were equal and very small, being from 1 to 1·5 mm. in diameter. It was very doubtful, moreover, if they reacted to light at all, or very sluggishly.

4th and 6th. Normal.

5th. Normal. Tactile sensibility and reaction to ammonia normal.

7th. Normal. Saliva secreted freely. Pressure on the sac caused also a copious lachrymal secretion.

8th, 9th, 10th, 11th, and 12th, appeared normal.

Spinal Cord.—Motion and sensation transmitted normally. All the visceral centres and reflexes were perfectly normal.

Convulsions.—It was found by Mr. R. J. Godlee, to whom the patient was first brought as an out-patient, that pressure on the solid mass into the interior of the sac caused a general convulsion. After its admission the following facts were observed:

Pressure on the right side of the peduncular process (passing through the hole in the occipital bone) gave rise to powerful contractions of all the muscles of the trunk and limbs, but most especially on the left side. In each group of muscles the sequence of events was the same, namely, clonic spasms passing into tonic contraction, this stage being often incomplete, with the existence of tremors as a result, the rate of the tremors being about that of ankle-clonus. The order of march of the spasms was face, upper limbs, trunk, and then the lower limbs.

Contraction of the upper group of facial muscles on both sides was first noticed, and this was followed by powerful contractions of the left lower facial muscles, the angle of the mouth on that side being drawn strongly upwards and to the left, while the nasolabial fold was very much exaggerated;

the right lower facial muscles also were seen to be contracting, but could not antagonise the left side. This is seen very well in instantaneous photographs (taken very kindly for me by Mr. Hyde Marriott) of the child while convulsed.

There were no movements of the eyes nor of the pupils following the contraction of the face; the upper limbs became flexed in all segments; the rigidity of the left, noted above, being exaggerated, and both limbs showing the tremor very markedly. As before said, most movement was on the left side (*i.e.* opposite to that pressed upon).

After the spasms had become fully developed in the upper limbs, they invaded the trunk muscles and caused flexion of the trunk, *i.e.* slight emprosthotonus, the movements being accompanied by powerful tonic contraction of all the muscles in the lower limbs, the result being that all the joints were fixed in a position of 90°.

Directly the pressure on the tumour mass was removed, the spasms left off, the limbs returning to rest after a few irregular movements. Pressure applied to the opposite side (the left) produced the same movements, but much less marked.

Simple pressure to the convex surface of the tumour caused feeble movements of the same kind.

Oct. 5.—In the afternoon of this day the sac was aspirated and 370 cc. drawn off. The chemical reactions, &c., will be given later, as they were almost identical with those shown by the fluid obtained from the tumour by subsequent aspirations. During the aspiration the child seemed absolutely unaffected in every way.

The intrasaccular portion of the brain could now be felt to be a bilobed mass with a rounded surface.

After the aspiration the tumour very slowly filled again, and by the end of seven weeks had recovered the size previous to aspiration, viz. circumference 16 inches.

It was believed that, as has often been recorded, a small process of the choroid plexus projected into the interior of the sac (this however was soon found to be erroneous), and therefore it occurred to me to try the effect of twisting the sac on its pedicle. This was easily done by marking quadrant lines on the sac, and by continuing them on to the head, a definite means of recording the amount of rotation was obtained. It was found possible to turn the tumour 45° without causing more than transitory uneasiness.

The rotation, however, had none of the desired effects of checking the refilling of the sac, which simply became more tense, and in three days the whole of the skin covering it was deeply congested and of a bright red colour.

The sac was therefore aspirated again, and 15 ounces with

drawn; but three days later the circumference was 14½ inches, and on the next day the child vomited. The sac was consequently turned back, when all these symptoms disappeared. On December 1st the sac was again aspirated, and 26 ounces drawn off. When the sac was completely emptied, the cerebral mass was felt to be distinctly fluctuating on the right side. A hypodermic needle, with syringe attached, was passed through the wall of the large sac into this smaller one, and 2·4 cc. of a slightly blood-stained fluid was drawn off. (This fluid was of a pale straw colour, was faintly alkaline, inodorous, and had a faint saltish taste. It gave no precipitate on boiling, but Millon's reagent threw down a cloudy precipitate, which turned brick-red on heating.)

During the following 24 hours, about 2 ounces of fluid collected in the sac. Twelve days later (December 12th), the sac was aspirated for the fourth time, and 17 ounces withdrawn.

The advisability of endeavouring to check the formation of fluid (see remarks) rendered it very necessary to know if possible what portion of the encephalon was protruded through the hole in the occipital bone. From a case[1] published at the time, it appeared very possible that the mass consisted of the corpora quadrigemina, and it occurred to me at once that this point could be cleared up, by applying the interrupted induced current to the tumour.

To the primary coil of an ordinary Dubois-Reymond coil with interrupter, a pint bichromate cell was attached, and small ball-pointed (¼ inch diameter) electrodes were fixed to the binding-screws of the secondary coil. The strength of current employed could just be borne by the fingers, the secondary coil being 4 cm. distant from the zero point (zero being the strongest). The following striking results were obtained.

(1.) When the current was directed *horizontally* through the mass, *no result* was obtained.

(2.) When one electrode was placed on the upper surface and the other on the lower surface, and the current consequently sent *vertically* through the hernia cerebri, there followed *rapid conjugate deviation* of both eyes towards the side stimulated. The conjugate deviation to the right, however, was not so marked as the movement to the left, which was very sudden. If the electrodes were taken off, the eyes returned slowly by jerks to the middle line again. If the electrodes were kept in apposition, the deviation of the eyes was not maintained for more than 10 seconds. Beyond salivation, the current caused no other symptoms, no change in

[1] Sutton, 'Pathological Transactions,' 1883, p. 18.

the pupil nor in the rigidity of the left arm. Patient showed no sense of discomfort from the current.

It was consequently assumed that the portion of brain involved was the corpora quadrigemina.

On December 20th the sac was aspirated, and 23 ounces of fluid drawn off; and again on December 28th, when 18 ounces were taken away.

In the hope of causing the sac to shrink, the posterior half was constricted off with a tape ligature, and into this portion was injected (December 28th)—with 18 minims of iodine solution—Pot. iod. gr. xx; iodine gr. x; glycerine ʒj.

The injection had no apparent effect on the child.

On December 30th, however, it was found that the wall of the sac was sloughing opposite the point of constriction, and therefore the much-delayed operation of cutting away the larger portion of the sac was commenced.

Although the patient was perfectly conscious, he did not exhibit any sense of discomfort when the harelip-pins used to temporarily close the sloughed opening were passed through the skin; consequently the following operation was performed without previously anæsthetising the child. As a precautionary measure, an india-rubber band was passed round the pedicle of the tumour to control hæmorrhage, but this was taken off almost directly.

A free vertical incision was made over the middle of the sac, the interior of which was found to be filled with a blood-stained serous fluid. In the posterior half of the sac the iodine had excited a little inflammatory disturbance, producing flakes of fibrine and soft trabeculæ, passing across the cavity, while the wall of the sac itself was œdematous and the surface hyperæmic.

The posterior half of the sac was then cut away freely with the knife, so as to leave two oval lateral flaps. It was now seen that the cavity was lined by the dura mater, in short, was part of the subdural space; for closing the aperture in the bone was a second much smaller sac, almost round, formed by a bluish membrane, and enclosing the hernial portion of the encephalon (see Fig.). It was clear that no good could result from opening this second sac, so an attempt was made to stop the "secreting" power of the dura mater lining the large sac, by sponging its surface over with chloride of zinc solution (40 grs. to ʒj). The line of incision was then closed with about thirty interrupted fine silk sutures; the wound was dressed with carbolic gauze and iodoform wool, the whole operation being performed under the spray. At the end of the operation, which lasted about 40 minutes, the child was very collapsed, unconscious, and cyanotic: 4 hours later, at 7 P.M., patient could

not swallow, so an enema of milk and brandy (ʒj) was administered. This revived the child so far, that by 3 A.M. it was warm (temperature 101°), and taking its food readily.

On the next day, January 1st, 1884, the sac was found to be half full of fluid, wound looking perfectly quiet; the next day the sac was found to be distended, so a small incision was made into the left side, and slightly turbid serum allowed to escape, the opening being kept patent with a slip of protective. During this and the next day the patient gradually got more drowsy, and refusing food, it died on January 4th. After the operation the temperature rose gradually in 12 hours to 101·8, it then varied from 99 to 103·2 being 103 one hour before death, and 103·4 a few minutes after.

Note on the General State of the Patient.—From the time of admission to its death it took its food very readily; but in spite of that, it steadily emaciated, the loss of fat (of which tissue there was but little on admission) being followed by wasting of the muscles. During the two months preceding its death the emaciation proceeded very rapidly, and for the last month the food passed per anum but slightly altered by digestion. Alteration in its diet made no obvious effect.

Analysis of the Fluid drawn off from the Dural Sac.

The general appearance of the fluid and its reactions were so constant, that one description will suffice for the various quantities.

The fluid was proved to be albuminous, and it did not contain sugar nor extractives.

Fluid was of a pale-straw colour, clear, watery, inodorous, and had a saltish taste. The reaction was weakly alkaline (fixed). There was no deposit, and sp. gr. = 1006—1015.

(1) Boiling produced a very faint cloud, insoluble in acetic acid.
(2) Addition of H_2O, nil.
(3) Addition of alcohol threw down a white precipitate partly redissolved in excess of H_2O.
(4) Addition of ·2 per cent. HCl, nil.
(6) Potassic ferrocyanide and acetic acid gave a dense, curdy precipitate, insoluble on boiling.
(7) Millon's reagent gave a pink precipitate, turning crimson red on heating.
(8) Silver nitrate gave a curdy white precipitate, soluble in ammonia.
(9) Boiling with Fehling's solution gave only a dirty brown precipitate.

(10) There were no extractives nor urea.
(11) Estimation of phosphates by uranic nitrate:

Date.	Quantity.	Percentage of phosphoric acid.
October 10th	370cc.	·011
November 25th	320cc.	·013
December 1st	728cc.	·017
„ 11th	476cc.	·02
„ 20th	644cc.	·028
„ 28th	504cc.	·028

NOTE.—It will be seen from this table of analyses, for which I am indebted to my friends, Mr. Hyde Marriott and Mr. MacCabe, that the proportional amount of phosphoric acid lost on each occasion gradually increased in amount as the case progressed, in fact, up to December 11th the increment was almost exactly ·003 per cent. This interesting fact should no doubt be considered in relation to the gradual emaciation of the patient; and the figures are here given in full, in hope that future observers may throw some light on the question.

Post-mortem, 26 hours after Death.—Rigor mortis almost passed off. Body extremely emaciated and very anæmic. The fingers of the left hand were still rigidly flexed on the palms, the thumb lying outside the forefinger. The sutured wound had healed perfectly by first intention, except just below the middle where a few stitches had been removed to improve the drainage. On laying the sac open it was found to contain a small quantity of sero-pus, while the sac wall itself was œdematous, being about ¼ inch thick, and its inner surface (dural) was acutely inflamed, and scattered over it were flakes of fibrinous exudation. The large sac was adherent to the inner arachnoidal one over the proximal third of its surface.

The arachnoidal sac projecting into the bottom of the dural cavity was tense, and while the upper part was of a yellowish colour over about one-fourth of the whole circumference, the remainder was dusky with a bluish-black tinge (post-mortem decomposition).

Skull.—The soft parts were now stripped off the skull, which was found to be apparently normal, exhibiting no true synostosis, the only unusual point about it being the size of the fontanelles, which were smaller than usual, but still preserved the ordinary appearances. The margins of the fontanelles, sutures, &c., appeared thick and pale, but afforded no evidence of congenital syphilis.

In the middle of the occipital crest there was an oval hole, through which the hernia had passed. This hole measured 1 inch long and ½ inch broad, covering almost one-third of the middle of the occipital ridge. The margin of the

hole was thin, being bevelled off on both surfaces, but its edge was smooth and round. Rest of skeleton perfectly normal.

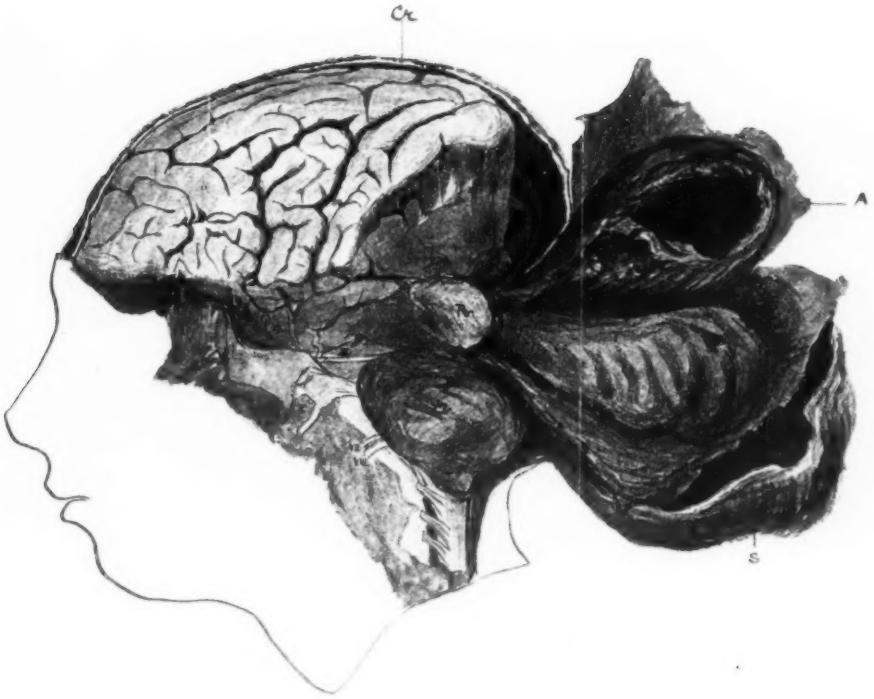
Meninges.—The dura mater was normal but thin, especially the tentorium. It was adherent by firm fibrous tissue to the margin of the hole in the occipital bone, and although thin, passed out to form the innermost layer of the sac wall, the tissue intervening between it and the skin being œdematous. The arachnoid also was quite normal, except at the margin of the hole, to which it was firmly united, and also to the dura, although the inner surface of this extra-cranial arachnoidal sac was covered with a layer of fibrinous exudation.

Sinuses.—Normal, except posteriorly, where the longitudinal divided at the apex of the lambdoid suture into two branches which ran down on either side of the occipital hole above described, and terminated in the lateral sinuses in the usual way, but opposite the middle of the cerebellum.

Central Nervous System.

The brain was exposed by cutting away the side of the skull and sac, as indicated in the figure, which is a photographic reproduction of a sepia drawing made life-size by careful measurements, and then reduced in the camera to two-thirds.

Cerebrum.—The cerebral cortex on the outer surface presented the normal fissures, but the secondary sulci were very numerous and complicated. Only the occipital lobe presented any peculiarity, and that lay in the fact that posteriorly the tips of both lobes were merged into the dilated corpora quadrigemina, the hernia cerebri. To make this clearer, it will be better to state at once, that on opening the arachnoidal sac of the encephalocele (before opening the skull) there appeared in view two ovoid masses, fixed by a narrow pedicle, which passed through the hole in the occipital bone to the encephalon. Of these two lobes, the superior, marked (N) in the figure, was a thin-walled sac formed of soft nerve-tissue, with a smooth fibrous exterior, and containing a turbid fluid with particles of degenerated tissue floating in it. This sacculated mass was found to be fixed to the pedicle by very firm connective tissue, continuous in front with the arachnoid and pia mater, which filled the sulci of the hemispheres, and helped to roof over the lateral ventricles, while posteriorly it wrapped round the pedicle of the rest of the encephalic mass (T), covering it, and continuous by its under surface with the sheet of tissue partly roofing the 4th ventricle. On vertical section this lobular mass (N) was found to exhibit, especially towards its fixed end, soft pink-grey tissue, somewhat resembling



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nerve-tissue. On microscopical examination, however, this was found to be exceedingly embryonic, consisting of neuroglia, with a few scattered small round corpuscles lying in distinct cell-spaces, forming a cortex, and beneath this was a layer of scarcely developed fibres.

The fibrous tissue forming the rest of the wall of this lobe was extremely vascular, evidently largely composed of pia mater, the vessels forming a rich plexus, mainly venous.

Possibly this mass represented the enormously enlarged pineal body, as it was only continuous with the rest of the cerebrum by the fibrous sheet, arachnoid, and pia mater above described; moreover, it fitted into the angle between the cerebral hemispheres and the corpora quadrigemina, and finally there was no other pineal body to be found. The inferior lobe of the hernia cerebri (T) proved to be the roof of the aqueduct of Sylvius bent backwards, so that the corpora quadrigemina were drawn out into the saccular form shown in the figure, the upper surface of this lower lobe being directly continuous with the posterior and inner aspects of the tips of the occipital lobes of the cerebrum. (In the figure this connection, together with the occipital and temporo-sphenoidal lobes of the left hemisphere, are cut away, so as to expose the Pulvinar. Pv.)

The sulci on the median aspect of the hemisphere were normal, except over the occipital lobe. Here, behind the parieto-occipital fissure, the sulci were arranged at right angles to the line of that fissure, and, running downwards and backwards, were lost among the fissures which marked the surface of the hernial lobe (T); shallow indications of these could be seen at the free extremity of the mass (see Fig.).

The aqueduct of Sylvius which formed the cavity of the lobe (T) was very narrow and flattened. On vertical section, the whole thickness of this lobe was clearly nerve-tissue in direct connection with the cortex of the occipital lobe of the brain, and on microscopical examination this lobe forming the corpora quadrigemina was found to consist wholly of nerve-tissue forming a cortex, with, underneath it, a layer of white fibres running forwards to the floor of the 3rd ventricle. This layer of course formed the wall of the central canal, and was covered on the inner surface by ependyma.

The Cortex (of the lobe T) presented characters intermediate between the structure of the cortex of the supra-marginal convolution (*vide* infra) and the slight remains of nerve-tissue found in lobe (N). Thus the basis consisted of a well-formed neuroglia, which in the medullary portion showed numerous round nuclei. In this ground-substance were scattered very numerous round corpuscles in cell-spaces. Immediately

beneath the outer surface of the lobe these became fewer in number, but there was no corpuscle-free layer as in the cerebral cortex (*vide* infra). The vessels in this part ran at right angles to the surface dipping in from the pia mater, and were accompanied by very narrow perivascular lymph spaces. All the vessels were engorged with blood.

The medullary portion was made up of horizontally directed fibres with parallel blood-vessels.

Microscopical Examination of the Cortex of the Left Supramarginal Gyrus.—The sections of this gyrus showed a perfectly normal (adult) condition of the fibres of the corona radiata, and also of the outermost layer of the cortex (*i.e.* submeningeal), so that further description of these parts would be superfluous. Of the intermediate part of the cortex (which was of the usual depth) there was the normal neuroglia, in which were scattered both angular and round nerve corpuscles, all varying in size from four to twelve times the diameter of a red blood disc. They were arranged in a definite layer immediately beneath the outermost submeningeal layer, but beyond a slightly marked similar arrangement just above the fibres of the corona there was no further indication of layers. The vascular supply and direction of the fibres was quite normal.

The Basal Ganglia presented normal appearances on section.

Commissures.—The corpus callosum was represented only by a thin layer of white nerve tissue roofing the lateral ventricles (*vide* also supra). None of the transverse commissures could be made out. The fornix was normally developed.

The Cerebellum consisted of lateral lobes, normally developed and united in front by middle peduncles (white) of fair size, but posteriorly the vermiform processes were solely represented by two lateral rounded masses which projected into the cavity of the 4th ventricle, and were only separated from one another by a thin layer of pia mater.

The 4th Ventricle.—Normally bounded in front, and at the sides, was covered posteriorly by a strong sheet of fibrous pia mater and arachnoid.

Spinal Cord.—The only abnormal appearance in the spinal cord was the persistent embryonic shape of the central canal. This in the cervical enlargement still presented the well-known isosceles triangular outline, with the basal ends of the sides bent so as to be almost perpendicular to the base. In the lumbar region the canal was an irregular trapezoid, directed obliquely across the antero-posterior axis of the cord. Throughout it was lined with the normal epithelium. In some sections from the lumbar region was noted a condition I have also seen in the adult, namely, a small process of neuroglia

usually opposite the posterior median fissure (pushing into the central canal for about ·5—1 mm. in depth. This process is not covered by the epithelium of the central canal.

With the exception of the alimentary tract, the viscera were normal, and there were no further developmental abnormalities.

Stomach.—Contained clotted milk, and abundant gastric secretion.

Intestines.—Both small and large were extremely pale, soft and thin. One large intestine contained clotted milk that was scarcely altered by digestion, only stained with bile.

Liver was pale, margins of lobules yellowish.

General Summary of the Post-mortem.—The death of the patient was clearly one from inanition due to the gradual failure of the alimentary tract to assimilate nutriment. The local inflammation in the sac of the tumour caused no symptoms beyond rise of temperature, and did not lead to general encephalitis. The outer sac had already adhered over the proximal third of the inner one, and the closed portion of the wound had healed by the first intention, so that local repair had proceeded very rapidly.

Remarks.

Beyond the minor point of interest, already referred to, in the composition of the fluid drawn from the sac, the questions of diagnosis and treatment alone call for attention.

Diagnosis.—The first case of occipital encephalocele which chiefly attracted notice to the subject was an observation of Lallement's,[1] on a patient aged 23 years, a young woman. In this instance, which was a small version of the one described above, the examination simply led to the discovery of a fluctuating tumour, and an exploratory incision was made into the sac, with the result of exposing an arachnoidal sac containing two lobes of the cerebellum. Although this was not opened, the patient succumbed in eight days to an attack of acute encephalitis and meningitis, no doubt of septic origin, judging from the brief but graphic history. This case illustrates very well the real difficulty of diagnosis in this condition, viz. (1) the presence or absence of a portion of the encephalon in the sac; and (2) the nature of the same. Obviously the former of these divisions of the question must be first determined, and supposing ordinary physical examination, such as palpation, translucency, &c., to fail, aspiration should be resorted to at once, the sac completely emptied, and the existence of a hernia cerebri determined. There should be no fear

[1] Richeraud, 'Nosographie Chirurgicale,' t. 2, pp. 396–322; 1815.

of awkward symptoms following aspiration. In the beginning of this century, Callisen[1] speaks of this trivial operation as quite safe, and in the case above reported, the only effect on the patient (even when the sac was absolutely emptied) was a very beneficial one. The lethargy which characterised it before usually disappeared at once, and the child crowed, moved its limbs and fed readily. (As a practical point it may be noted that the wall of the sac being inelastic, the fluid tends to ooze through the aspiration puncture, even when covered with collodion. It will be found that a fine harelip-pin and a loop of carbolised silk meets the occasion, by stopping the flow and healing the aperture.) With regard now to the position of the brain herniated, it is clear that in event of failure of ossification, in some portion of the occipital bone we may have protruded either the cerebellum,[2] the corpora quadrigemina,[3] or the occipital lobes,[4] all three together, on any combination of two, this being the commonest condition.

Under these circumstances the position of the tumour will offer some guide to finding the nature of the hernia by observing the relation of the tumour to the tentorum cerebelli. When beneath this structure, the hernia is practically certainly cerebellar;[5] when opposite to it, as in this case, the protruded mass may be the corpora quadrigemina; and when above, then the posterior extremities of the occipital lobes.[6]

It is often, however, not easy to make out the exact position of the pedicle of the mass involved in the sac, and so deductions have been drawn from motor symptoms, either chronic or excited by interfering with the tumour. Thus, for instance, in several cases,[7] as in the present one, there has been noted tonic contraction of the flexor muscles of one or the other upper limbs. In my case it was the left which first passed into this state, and very much later near death the right became similarly affected. In Lawrence's case just quoted, the sides were reversed, but the march of the rigidity was very much the same. This unilateral (in commencement, at any rate) condition may perhaps be due to irritation of the crura from the tension produced by the dragging backwards of the tumour. That this occurs was beautifully seen on the inner aspect of the cerebral hemispheres in the present case, where, as described above, the sulci are directed along the lines of the tension. This

[1] 'Chirurgicæ Hodiernæ,' vol. ii. pp. 512–514; 1800.
[2] Richerand, loc. cit.
[3] Sutton, 'Pathological Transactions,' 1883, p. 18.
[4] Breschet, Obs. xxii., 'Archives générales de Médecine,' 1831, t. 25 and 26, p. 452 and p. 74.
[5] Breschet, op. cit., Obs. xiii., xxvi.
[6] Ibid. Obs. xxii., xxii., xxiv.
[7] Ibid, Obs. xiii.; also Lawrence, 'Med. Chir. Transact.' 1857, p. 307.

harmonises too with what is known concerning the mode of origin of this condition (*vide* infra).

A more equivocal symptom is the convulsive stage which can be elicited by pressure on the whole tumour, or better (*vide ante*) on the side of the hernia cerebri. In a case reported by Wedemeier,[1] pressure caused loss of consciousness as well as convulsions; while Bruns[2] describes in addition, turning of the eyes, deep respirations, weakening of the pulse, sopor, twitchings, vomiting and choking; all these symptoms disappearing immediately the pressure is removed. From the detailed description given before of the similar phenomena as observed in the present case, and from the quotations just given, it is obvious that very little aid can be expected from such symptoms to help in localising the portion of brain protruded. This opinion is also supported by an observation by Job à Meeckren[3], who described a case in which the patient was two years old, and in which pressure on the sac caused syncope and loss of sight. It was found post-mortem that the dura mater (*sic*) closed the foramen in the skull.

The very general nature of the symptoms points to their finding origin in merely sudden increase of encephalic pressure, and in the present case this was proved to be so by squeezing only the distal portions of the sac, when the same phenomena were elicited, but at the same time not to so marked an extent.

Appreciating fully then the uselessness of mechanical stimulation, there remains electrical, and provided that in any particular case the efferent fibres from the hernial portion of the encephalon are intact, and the said portion well developed, we have, I believe, in the induced current an adequate means of diagnosing the part involved.

We might expect that stimulation of the corpora quadrigemina would always elicit movements of the eyes, while stimulation of the median portion of the cerebellum would produce in addition to movements of the eyes extension of the neck, &c., and irritation of the occipital lobes probably nil. In the present case only the eyes moved to the same side as that stimulated, and nothing else occurred. This was noted by Ferrier[4] as a result of irritating the pyramid of the median lobe of the cerebellum in monkeys, but in addition there ensued movements of the head and neck. That stimulation of the corpora quadrigemina produces movements of the head as

[1] Wedemeier, 'Journal für Chirurgie u. Augenheilkunde,' 1826, Bd. 9, p. 152.
[2] Bruns, 'Die Chirurgischen Krankheiten in Verletzungen des Gehirns,' &c., 1854, pp. 695–720.
[3] Quoted by Ferrand, 'Mémoires de l'Acad. de Chir.' t. 5, p. 863; T. 13, p. 102.
[4] 'Functions of the Brain,' 1876, p. 98.

well as the eyes, is of course well known, but that movement is in the same direction as that taken by the eyeballs, and not at right angles as in the case of the cerebellum. However, of course, but little stress can as yet be laid upon this point, which is only brought forward to suggest an additional means of diagnosis.

Treatment.—The treatment adopted in this case was that indicated by Callisen (loc. cit.), with the additional attempt to diminish the secretion of fluid by twisting the vessels of the pedicle.

It is perfectly clear that aspiration per se is of no use, and aspiration followed by pressure is equally futile. To arrive at some definite course of rational treatment, we must for a moment consider the probable mechanism of the affection. Seeing that we find every stage between meningocele and encephalocele, from the slight bulging of a fontanelle to the most complicated palatine hydrencephalocele,[1] it is no wonder that Adams[2] in 1883 suggested that the first stage consisted in a meningeal protrusion, the pressure in which was kept up by increased secretion within, and that the cerebellum followed, half pushed, half dragged out. The theory of adhesion in utero to the amnion at a very early period, evidence of which is found by those supporting it, in the scars observed on the cutaneous surface of the sac (*vide antea*), accounts readily for the primary production of the cranial defect, and the more so, since, as in the present case, there is not always premature synostosis of the sutures. Of course when the latter malformation occurred[3] it would form a strong predisposing cause. Doubtless in different cases, each one of these causes or combinations of them may be in operation.

Treatment clearly then should be, first to stop, if possible, the meningeal hernia, and the first successful case[4] was treated by pressure applied by means of a leaden plate. The tumour in this instance was very small, and easily dealt with, but in more severe cases, as in the above, it would obviously be quite incompetent. Since the sac formed by dura mater and arachnoid continues to secrete serous fluid and yet the lymphatics running towards the base of the skull are blocked in the pedicle of the tumour (by simple pressure at first, at any rate), it is obvious that the natural direction (as I regard it) of the circulation of the cerebrospinal fluid is interrupted, and therefore must collect in the closed sac, producing all the symptoms resulting from pressure on the encephalon. The only course

[1] Virchow, 'Die Krankhaften Geschwulste,' 1863, p. 188 et seq.
[2] Adams, 'Dublin Journal,' 1833.
[3] Breschet, loc. cit., Obs. xv.
[4] Salleneuve, quoted by Ferrand, q. v.

open to relieve this state of things would appear to be this: cut away the posterior half of the sac, remove the dura matral lining of the peripheral portion by rapid dissection (the application of strong antiseptics caused, I think, too much inflammation of the sac in my case), and bringing the edges together in the middle line by a continuous suture of chromic catgut. Drainage must of course be provided for, by a free opening at the lowest point. By the employment of continuous suture, I am confident the operation could be performed in fifteen minutes, and shock thus greatly avoided. Of course it must be carried out with the strictest antiseptic precautions, as the mortality of septic meningitis makes perfectly clear the folly of opening the dura mater except under those conditions.

The sac when healed would have united firmly over the arachnoidal sac covering the encephalocele (as it was rapidly doing in the present case even when the patient could scarcely be said to be assimilating any nutriment). When healed in this way, no further secretion would occur, pressure would prevent further hernia cerebri, and adhesions fix everything in situ.

It is perhaps not chimerical to suggest, that a bony covering could be obtained for the tumour by transplantation of periosteum, &c.

The fact that of 44 cases collected by Lawrence the cerebrum was normal only in 5, and further, that all the recorded cases I have met with where the patients attained to an age of discretion were imbeciles, renders the successful application of treatment doubtless somewhat dispiriting; still, that of course should not weigh in discussing the merits or demerits of any particular line to be adopted.

NOTES ON A CASE OF PURE APOPLECTIC BULBAR PARALYSIS.

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Apoplectic bulbar paralysis, when associated with hemiplegia, is not of unfrequent occurrence; but when the bulbar symptoms alone exist, the case, from its rarity, is of exceptional interest. The reason is obvious: the anatomical relationship of many of the motor nuclei in the medulla is so close, that an extremely localised lesion is required in order to limit the mischief to two or three motor nerves. Apoplectic bulbar paralysis is usually associated with alternate hemiplegia, or with paralysis of the upper, lower, or of all four extremities. The disease, as a rule, is initiated by an apoplectic seizure, varying from a momentary loss of consciousness to a profound attack, which in some cases is instantaneously fatal. The region attacked is perilous, and very little deviation of the lethal force is sufficient to convert a circumscribed lesion, capable only of producing a local paralysis of a limited extent, into an occasion of immediate death. In looking over the literature of the subject I have been able to find only three cases comparable with the one about to be described, as regards the limited area attacked, and the uncomplicated nature of the onset of the disease.

In 'Guy's Hospital Reports for 1870,' Dr. Wilks mentions a case in which the patient, whilst at dinner, fell off her chair without losing consciousness, and at the same moment lost the power of speech. She could not swallow without great difficulty, and had constant dribbling of saliva from the mouth; the mental faculties and powers of locomotion were undisturbed. The patient lived for two or three years after the attack, but never spoke again. Hérard, in the 'Union Médicale,' 1868, reports the case of a man aged 68 who was healthy up to the time of the attack. He awoke one night and found himself unable to speak; both tongue and lips were paralysed. The left corner of the mouth was lower than the right; food fell out of the mouth, and saliva constantly ran

over the lower lip. The act of swallowing was not greatly interfered with, but the movements of the vocal cords were imperfectly performed. The intelligence was unaffected, and the limbs retained their normal power. The patient gradually improved, and was able to resume his work, although nine months after the attack a trace of the injury still remained.

A third case by Charcot is reported by Huret in his 'Tribut à l'histoire de l'Embolie des artères vertébrales, 1873.' It is that of a woman aged 68, who, three years before the attack in question, had an attack of left hemiplegia from which she had quite recovered. One day, without any apoplectic seizure, she lost her speech; she understood all questions quite well, but could not answer them. The right corner of the mouth hung down and could not be moved; the movements of the tongue were partially lost. The patient had considerable difficulty in swallowing, the food returning partly through the nose. There was no paralysis of the extremities. The right pupil was smaller than the left; the heart-sounds were normal. The symptoms gradually improved, and in three months the patient could almost be said to have recovered; the right corner of the mouth, however, remained somewhat lower than the left. In addition to the above cases, Duret quotes one of Voisin's in which bulbar paralysis occurred suddenly, but I have not been able to meet with a detailed account of the symptoms. The following case came under my own observation.

M. D., a married woman, 42 years of age, rather above the average build, of pallid complexion, active in habits, and free from any syphilitic taint, eleven years ago had an attack of rheumatic fever. Since the fever, she has, on two occasions at least, suffered from general dropsy of cardiac origin; at the time of the attack about to be described, she was in her ordinary health. She had not previously suffered from any cerebral symptoms. The patient attributed the attack to a state of excitement (induced by some domestic trouble), into which she fell shortly before the seizure.

On the 27th of October, 1883, whilst walking in the street with another woman, the patient suddenly became conscious of having lost the power of speech. This occurred without any apoplectic seizure whatever; she did not fall, nor had she any pain or dizziness in the head, nor did she even momentarily lose consciousness. Within an hour after the occurrence of the symptoms, the patient walked without difficulty to the Salford Royal Hospital, a distance of about half-a-mile, and there saw Dr. Haslam, who has since informed me that her mental faculties were then quite clear, and that her bodily movements, except those of the tongue and mouth, were perfect. She went home, and remained there for a few days, when, on account of the difficulty experienced in

administering nourishment, she was admitted into the hospital, and came under my care. On November 1st, 1883, her condition was as follows:—Mental faculties quite clear; she responded intelligently to questions either by signs or by writing on the slate. The tongue, somewhat coated, lay flaccid on the floor of the mouth, and could not be moved in the slightest degree, by any effort of the patient. The soft palate was flabby, and hung loosely down, bringing the pillars of the fauces somewhat forwards. The pterygoids were completely paralysed, no lateral movement of the lower jaw being possible. The masseters and buccinators did not appear to have suffered so much; the jaws could be brought together, but with less than the usual power. The muscles attached to the lower jaw and the hyoid bone were not affected, the elevation of the larynx in the act of deglutition being normal in amount. The orbicularis oris was completely paralysed; the patient was totally unable to purse the lips as in the act of whistling, nor could she kiss the back of her hand, nor blow her nose; the latter disability, of course, was partly due to the condition of the tongue and soft palate, as well as to that of the orbicularis oris. The levator anguli oris, levator labii superioris, and zygomatici of the right side were completely paralysed; the corresponding muscles on the left side were not fully paralysed, but their motility was very slight; the patient was only able to impart the least flicker of movement to the left corner of the mouth, and none whatever to the right. The corners of the mouth were equal in height. The ocular and upper facial muscles were altogether unaffected; the patient was able to close the eyelids, and to resist any attempt to open them by force. The trapezius, sterno-mastoid, and all the other muscles of the trunk and extremities were altogether unaffected. The nerves of special sensation were severally tested, and found to be in their normal condition. There was no singing in the ears, nor was there any sensory paralysis. All the facial muscles responded normally to both constant and interrupted currents; the reflex of the soft palate was not materially, if at all, lessened. The only sound the patient could utter was a nasal "a," being the primitive sound produced by the vocal cords without glossal or labial modification. A laryngoscopic examination revealed nothing abnormal about the action of the vocal cords. Saliva ran continuously out of the mouth, but there did not appear to be any excess in amount; the overflow was simply the result of inability to swallow, and to close the lips. On being asked to swallow, the patient took some water into the mouth, and after a momentary pause, accompanied by a peculiar, expectant look, she slowly turned back her head so as to allow the water to flow towards the fauces. The result was an explosive cough, which forcibly ejected the fluid both from mouth and nostrils. Occasionally the attempt was more successful, and the fluid wholly or in part reached the œsophagus; the act of swallowing, however, was always followed by more or less cough. It was observed that in the early stage of the disease the patient could swallow aërated fluids with greater chance of success than

still fluids. The temperature was normal, the pulse 72; there was no increase in arterial tension. A slight mitral systolic murmur was heard over a limited area. The urine contained neither albumen nor sugar.

For the first day or two after admission the patient was nourished by nutrient enemata, afterwards she was fed with the œsophageal tube. On the eighth day after admission the tongue had recovered a slight amount of motility, and as the patient strongly objected to the use of the tube, she was allowed to try to swallow small quantities of finely divided meat. When she concentrated her attention on the act of swallowing, and performed it in a deliberate manner, as a rule she was successful; but if she attempted to swallow quickly, the food was promptly ejected from the mouth by an explosive cough. Fourteen days after the attack she could protrude the tongue about a quarter of an inch beyond the teeth, could slightly purse the lips, and swallow prepared food with tolerable certainty; she was, however, quite unable to masticate. She has continued to improve, and her present condition, six months after the commencement of the attack, is as follows:—The tongue can be protruded about half an inch beyond the teeth, but she cannot lift it to the roof of the mouth, nor can she curl it up at the sides. She can purse the lips pretty well, but without much power; she cannot smack them, nor can she blow her nose. She can swallow soft pulpy food better than fluids, but cannot masticate even bread. There is now a trace of lateral movement in the lower jaw, and the muscles on the left side of the face have considerably improved; the corresponding muscles on the right side have also recovered a limited amount of motility. The patient can make some attempt at articulation, and occasionally one can make out the meaning of what she endeavours to say, but there is no real speech. She complained a few week ago of giddiness when walking, but has improved in this respect. She also complains of weakness of vision. In an early stage of the case I examined the discs without finding anything abnormal, at that period the patient made no complaint about her vision. Later on, my friend Dr. Glascott kindly examined them and found the retinal veins engorged, especially those of the right side, the margins of the discs were ill-defined, but there was no retinitis. The deep reflexes of the arms are now slightly more marked than in the normal state; in the early stage nothing abnormal in this respect was observed. The compressive power of the hands tested by the dynamometer equals 25 kilogrammes for the right, and 23 for the left; the difference is too slight for unilateral paresis, and is more probably due to the usual predominance in muscular power of the right hand over the left. The excess of reflex just mentioned would probably be caused by slight inflammatory processes set up in the immediate neighbourhood of the focus of injury; the disturbance of the retinal circulation might proceed from the same cause.

There is not much difficulty in localising a lesion such as

this, the symptoms at once point to the posterior part of the rhomboid sinus—to a region embracing the nuclei of the hypoglossal, facial, and motor branch of the fifth nerves. The facial nucleus proper is removed from that of the hypoglossal; but the inferior facial nucleus is about on a level with, and closely external to it. It is supposed that the accessory facial nucleus exclusively controls those branches of the facial nerve which supply the lower facial muscles: it is therefore probable that, as regards the facial nuclei, the accessory nucleus only was attacked, the supply to the upper facial muscles on this account not being interfered with.

As regards the nature of the lesion, acute myelitis may be omitted, as this disease, although it may occur very suddenly, requires at least several hours to develop, and usually runs a rapidly fatal course. The present case, on the contrary, was instantaneous in onset, and the tendency from the first was to improvement. There is then left the choice between blood-extravasation, thrombosis, and embolism. Against extravasation is the early improvement, and the improbability of the occurrence of such a very limited effusion. Whatever was the cause of the lesion, it was of a peculiarly circumscribed and selective character; it attacked a series of nuclei in the same order that they are attacked in progressive bulbar paralysis, the course of which follows a certain definite sequence. It is difficult to imagine that extravasation—the amount and localisation of which would be determined by contingency—could produce such a methodic arrangement of symptoms. It would rather be expected that other motor centres would be attacked, and that an anomalous chain of symptoms would result; still the rupture of a miliary aneurism might produce a sufficiently localised extravasation. Thrombosis presupposes, more or less, the existence of preliminary symptoms: the gradual closure of a vessel would reduce the blood-supply of the region nourished by the vessel, and would probably produce prodromata as dizziness or paræsthesiæ, both of which were absent in this case. Here again the elimination cannot be absolute, for thrombosis does at times develop symptoms suddenly and without previous indications. Embolism seems to be the most likely causation: the attack of rheumatic fever and the subsequent heart mischief give a certain colour to this supposition. It is probable that an excited action of the heart, induced by the domestic trouble before mentioned, led to detachment of a minute vegetation from the left side of the heart, which was carried with the blood stream along the vertebral and basilar arteries and finally lodged in one of the small branches. Almost all the arteries of the medulla are terminal arteries, and therefore such a

clot would give rise to one or more of those well-known wedged-shaped infarcts, with their basis directed towards the floor of the fourth ventricle, and including in their sphere of action the nuclei before mentioned.

There is one point worthy of note—the two groups of muscles most profoundly attacked in this case, the same muscles which are first attacked in progressive bulbar paralysis, viz. the extrinsic lingual muscles and the orbicularis oris, were the first to show signs of returning motility, and have relatively undergone the most marked improvement. The nuclei concerned are the accessory facial and the hypoglossal: the hypoglossal nucleus receives its blood-supply from terminal branches of the anterior root-artery and of the anterior lateral artery. Both the hypoglossal and the inferior facial nuclei are supplied by branches from the artery of the median raphé. Assuming embolism to have been the cause of the paralysis, it is possible that the main blood-supply of the nuclei affected was cut off by the embolus, producing for a time total cessation of function, which was afterwards restored within certain limits by collateral supply.

CASES OF CHOREA.

BY W. B. HADDEN, M.D.

A. *Complete loss of Articulation in Chorea.*

Case I.—A. L. æt. 7, female, was admitted into St. Thomas's Hospital, under Dr. Bristowe, on January 10th, 1879. She had never had acute rheumatism. Eight months before admission she had been under treatment on the surgical side for chronic abscesses around the knee-joint. Her present illness began two weeks ago, with movements chiefly affecting the right side. On admission, the chorea was found to be general, but most marked in the face, tongue, and right side. There was great difficulty in articulation, and also some dysphagia. She had tinea tarsi on the right side, and several chronic indolent ulcers on right leg and thigh, apparently the result of bone disease. No cardiac murmur was detected. On January 19th it was found that she could not speak. Next day she made an attempt, said "yes" and "no," but refused her name. On January 22nd she had recovered speech, but the words were jerked out, and syllables often mispronounced and slurred. The day following she was very quiet, and speech was again entirely lost. The muscles of the face were less affected, and the tongue protruded more easily. The temperature the previous evening was 102°·2. On January 24th, that is two days later, she could say a good many words quite distinctly, protruded her tongue quietly, and was altogether better. On February 3rd she was again unable to articulate, and the movements of the legs were markedly choreic. On February 18th, she could say several words distinctly, and repeat her name. From that time she steadily improved, and was discharged cured on March 19th.

Case II.—S. G., æt. 14, female, was admitted under Dr. Stone, on May 27th, 1879. She had never had acute rheumatism, but there was a history of fright just before present attack. On admission, she had general, but not very violent, choreic movements. The speech was slow, but not otherwise affected. She could not feed herself, and swallowed with difficulty. On

June 26th she was found to have lost all power of articulation, and this continued for more than a week. I found that the right hand was decidedly weak, and that there was anæsthesia of left arm and slightly of left leg. There was a soft systolic murmur at the apex of heart. She was discharged relieved on August 6th.

Case III.—S. R., æt. 12, female, was admitted under Dr. Harley, on May 30th, 1883. She had had acute rheumatism when six years old. In January, 1883, she had another attack which was very severe, and was followed by chorea and loss of speech. On admission, the movements were general, but most marked on right side. She could not articulate, but the next day she said a few words. On June 14th it was noted that the patient not only does not talk, but makes no attempt to frame words, and did not even try to speak to her mother. She protruded the tongue when asked. Since the previous day she had screamed much, and was very restless. On June 16th she could talk a little, and from that time continued to improve. There was a systolic murmur at the apex. She was discharged cured, on July 19th.

Remarks.—It is a curious and suggestive fact that, in all the cases the choreic movements were most marked on the right side, and in one that there was decided paresis of the right arm. The inability to articulate seems comparable to the paralysis of limbs, which may either precede or follow an attack of chorea. But whether the actual cause lay in the brain itself, or in the medulla, or in the exhausted muscle, I am not prepared to say.

B. *Anæsthesia in Chorea.*

Case I.—E. M., æt. 20, female, was admitted under Dr. Harley on May 6th, 1879. She had had convulsions during dentition, and acute rheumatism ten years ago. Six weeks ago she had a severe fall, and was much frightened. Three days later, movements began in the left arm, and were limited to that part until ten days before admission, when the chorea became general. About this time she was said to have had another fright, and since then her memory has become impaired, and she has had delusions. On admission, there were general choreic movements, but the left side was much more affected than the right. The left arm and leg, too, were very weak. The speech was but little impaired, and there was no difficulty in deglutition. These was total anæsthesia of left arm and leg, and partial anæsthesia of right arm. Her mental condition was much impaired. She did not know how long

she had been in the hospital, but she answered readily when spoken to, and had no delusions. There was no cardiac affection. On May 9th she became very restless and violent. She now had distinct delusions. She thought she was going to be poisoned, and that she would never see her parents again. At this time she was being treated with conium. On May 28th she developed well-marked symptoms of conium-poisoning, and accordingly the drug was omitted. The same treatment was subsequently resumed, but without benefit. On June 22nd she tried to drink a bottle of carbolic acid, but was fortunately observed. On July 9th, however, she was found with an empty bottle, labelled "poison," which had previously contained ʒ vij of a solution consisting of morph. acet. gr. $\frac{1}{4}$, and atropiæ sulph. gr. $\frac{1}{60}$ in ten minims. It was not until two hours and a half later that any symptoms were observed. It was then noticed that she tottered when walking. She soon went to sleep, and four hours after the solution was taken, she was apparently sleeping quietly. Half an hour later she was heard to groan feebly, the lips were blue, and the respiration seemed to have ceased. The extremities were very cold, the pupils dilated, the conjunctivæ insensible. Artificial respiration was kept up for twenty-six hours, and stimulants administered. No spontaneous effort at breathing was observed for twenty-four hours. She was comatose throughout, but could easily be aroused by shaking. Her mental condition was more unsatisfactory than before. She subsequently acknowledged to taking the poison, but said she poured some away. She was discharged on July 22nd, and I believe went to an asylum.

Case II.—E. R., æt. 15, female, was admitted under Dr. Ord on Jan. 22nd, 1879. Her mother and eldest sister were liable to faints, but there was no decided neurotic or other taint in the family. The patient had never had acute rheumatism or scarlet fever, but for the last six months she had been under treatment for enlarged glands in the neck. A month ago she began to have twitching of the left hand, then the face became affected, and lastly the leg. On admission, there were slight choreic movements of the head, left arm and both legs. There was also paresis of the left arm. It was found that there was anæsthesia of the left arm and leg. A systolic murmur was audible over the pulmonary artery. She was discharged cured on March 19th.

Case III.—E. B., æt. 16, female, was admitted under Dr. Bristowe on June 25th, 1879. She had had rheumatic fever, and five-and-a-half years ago was attacked with chorea. She had a second attack three months later, and from that time until May 1st, 1879, there were occasional tremblings of the

hands, feet, and face. On that day she had a fresh attack of rheumatism, which was followed by chorea. The movements affected both hands and feet, and slightly the face. The power of the right hand was much impaired, although the movements were less evident here than on the opposite side. She did not feel when a needle was thrust into the right leg, but flinched when touched with a pin on the left leg. Her mental condition was fairly good, but occasionally she called out in her sleep. There was a mitral systolic murmur. On July 8th, sensation began to return in right leg, and on the 18th, there was only a little difference between the two sides. She was discharged cured on July 27th.

Remarks.—I am inclined to think that there is no rule as regards the distribution of the anæsthesia in chorea. In Cases I. and II., as well as in two or three others which I have observed, the impairment of sensation was on the side most affected by the movements. In Case III., however, it will be seen that the anæsthesia was chiefly in the right side, whereas the choreic movements were mainly left-sided.

C. *Hysteroid Convulsive Attacks in Chorea.*

C. W., æt. 15, male, was admitted under Dr. Bristowe on January 16th, 1882. On December 20th he suddenly became faint and sick, and fell on his face. He was helped up, but a few minutes later he fell again, and began to kick about. He had some half-dozen such attacks on the same day. The choreic movements are said to date from this time. The boy was found in the casualty room, lying on a couch, with the body rigid, and working his arms and legs about. He was quite conscious, but emotional, crying readily when asked about himself. His mother said that before the fit he complained of not being able to swallow, and of tightness in the throat. Later, he was found to have characteristically choreic movements, his speech was imperfect, there was no anæsthesia, and no heart affection. On January 22nd he complained of a lump in his throat, and very soon he jumped up, and would have fallen out of bed if he had not been caught. He rolled over on his face, sobbing. When he was turned over, his back became quite rigid, and he worked his limbs about. He did not lose consciousness. He was discharged cured on January 31st.

Remarks.—Emotional disturbance is common enough in chorea, but the hysteroid convulsive attacks which occurred in this boy are probably rare. Unfortunately there is no record of the family history, nor of the previous history of the condition of the patient.

A CASE OF DORSAL PACHYMENINGITIS.

BY HOWARD H. TOOTH, M.B.,

Assistant-Physician to the Metropolitan Free Hospital.

THE case about to be related, though somewhat deficient in clinical details, yet is possessed of some pathological interest.

The patient G.G., aged 60, a compositor, was admitted into one of the surgical wards of St. Bartholomew's Hospital, on October 14, 1883, suffering from simple fracture of the lower third of the left femur, owing to the accident of falling downstairs.

The notes of the case as regards spinal symptoms are unfortunately rather meagre. They may be summarised as follows.

For ten years at least he has suffered from progressive paresis of lower extremities, which increased to such a degree that for five years he has been unable to get about without assistance. During this period and till death, has been subject to excruciating shooting-pains in the lower limbs, most severe in the left. There were also frequent pains of the same character in the left side, about the position of the lower ribs. No atrophy of muscles. Great reflex excitability of both legs, with sudden painful spasms at night for some years. Bowels always very costive, but no loss of control over rectum. Has had to use a No. 10 catheter for over five years, unable to pass water without it. His wife says that he has been extremely irritable and capricious in temper lately. He is the father of five children, two healthy, three died at about fifteen months "of convulsions and teething."

No history of syphilis, direct or indirect, could be elicited.

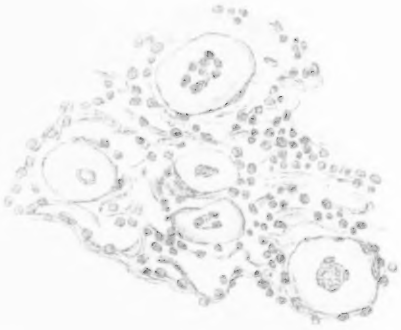
Oct. 14*th*, 1883.—On admission, considerable rigidity of both legs was noted, most obvious in the sound limb. Thigh adducted. Intense reflex excitability; so much so in the fractured limb, that splints had to be discontinued and the limb laid on a pillow. Distinct loss of sensation in both limbs. Upper extremities quite natural.

Oct. 19*th*.—A large bedsore discovered over sacrum on right

1.

3.

2



4.

side. Cystitis. Urine highly coloured, offensive; sp. gr. 1020. Alkaline. Albumen and pus.

Oct. 20th.—Pain in right shoulder, with some difficulty in raising the arm.

Oct. 23rd.—Shoulder better.

Oct. 25th.—Cystitis improved. Diarrhœa.

Nov. 14th.—Bedsores over both great trochanters.

Nov. 30th.—Abscess in scrotum. Opened.

Dec. 28th.—Going on well till this morning, when he had a rigor at 6 A.M. Temp. 103·4°.

Jan. 13th, 1884.—Another rigor at 6 A.M. Temp. 102·6°; unconscious muttering, delirium.

Jan. 31st.—Sore on back worse. Edges undermined, spines bare. Right great trochanter exposed.

Feb. 11th.—Died. The temperature during this period was always above normal at night, sometimes 103°–104°, often above normal in the morning.

At the autopsy, made on Feb. 12 by the surgical registrar, Mr. Bowlby, to whom I am indebted for the spinal cord, the cadaver was found to be well nourished. No atrophy of muscles. Brain quite natural. Thoracic organs natural. Liver, spleen, stomach and intestines quite natural. No signs of visceral syphilis. Kidneys enlarged, and suppurating in places. Ureters dilated, and bladder much thickened.

Spinal Cord, Macroscopic appearances.—Whole arachnoid-cavity much distended with clear fluid. At about the middle of the dorsal region, involving a length of about 7 cm., the membranes are fused together into a tough white leathery mass, about 5 mm. thick. This thickening involves rather more than one-third of the circumference of the sheath, and is adherent closely to the cord along the posterior half of its circumference. The cord at this point is much flattened out, measuring only 5·5 mm. antero-posteriorly. Here also the cord is twisted and distorted, being drawn round from right to left, so that the thickened mass, though adherent over the posterior segment, lies in the bony canal rather to the left side. It is not adherent to the bony canal, nor is there any disease of the vertebræ. The posterior roots are involved in the membranes on both sides. Anterior roots natural.

Microscopic examination.—Cord hardened in ammonium bichromate solution for three weeks. Stained in picrocarmine and hæmatoxylin.

Mid-dorsal region (Fig. 1). *Membranes*—Thickened dura mater composes about one-fifth of the whole mass. The component bundles of connective tissue are separated by large numbers of leucocytes. Vessels are numerous, the walls of which are not markedly thickened. Between the dura and

pia mater is a mass of fibrous material making up quite $\frac{4}{5}$ of the whole. This consists in its outer $\frac{3}{4}$ of bundles of large wavy structureless fibres, cut in all directions, but mostly running parallel to the fibres of the dura. There are numerous lacunæ containing nuclei, but no vessels. As the inner boundary is reached, the fibres become smaller and the lacunæ larger, containing numbers of leucocytes. Here numbers of blood-vessels make their appearance (Fig. 4). The vessels here, and to a somewhat less degree in the succeeding sections of the cord, have remarkably thickened hyaline walls. All distinction between middle and outer coats is lost. The elastic lamina is undistinguishable in most of the sections. In some the intima is considerably thickened. There is in some of the vessels a distinct proliferation of nuclei of the intima.

Cord.—Flattened antero-posteriorly. Right half smaller than left. Relation of parts distorted, as seen in figure.

In the white matter, the whole section shows softening with destruction of nerve fibres. The softening is much more marked on the anterior or unattached segment than on the posterior adherent segment. This must be due to latter being in a measure protected from the acute inflammatory processes spreading in from the spinal canal. The thickened vessels pass into the cord from the pia mater, and are scattered plentifully in cross-section over the whole section.

The right anterior horn of the grey matter is much attenuated; only one or two ganglion cells can be found, and they are highly degenerated. Left anterior horn also greatly affected, though less so than right. Some four or five ganglion cells are visible, but all showing more or less granular degeneration. Left posterior horn elongated and attenuated to a much greater degree than the right. The left posterior roots show in section proliferation of nuclei with considerable disorganisation of the nerve fibres. The right posterior roots look natural. Both show great hyaline thickening of the vessels, which in this part reach their highest degree of degeneration.

Upper dorsal region (Fig. 3).—Fibrinous thickening of pia mater. Marked softening of the white matter all around the periphery of the section, with complete disappearance of nerve tubules in the softened area, except on each side of the anterior fissure.

As the grey matter is approached, the nerve fibres make their appearance, and here the neuroglia becomes considerably thickened and condensed. This is most marked in the posterior columns and reaches its greatest degree in the postero-median columns, where there is considerable sclerosis. Even here there are, however, many axis-cylinders. The ganglion

cells of the anterior horns are wanting in sharpness and distinctness.

Cervical region.—To the naked eye the carmine-stained sections show slight deepening of colour marking the postero-median columns, and under the microscope there is a marked increase of the neuroglia. Except for the peripheral softening, as in the preceding section, the cord is natural here.

Upper lumbar region (Fig. 2).—Shows a considerable increase of connective tissue in the posterior columns. This is most marked in the middle $\frac{1}{3}$ of the posterior radicular zones, where large branching fibres pass from the posterior horns towards the posterior median fissure. The white matter between this and the transverse commissure shows similar connective tissue-increase, but to a less degree. The same may be said of the lateral columns close to the grey matter.

Sections taken lower still in the cord show less and less departure from the normal.

Remarks.—In spite of the lack of syphilitic history, there can, I think, be no doubt of the specific nature of the affection in this case. It probably originated at a gumma of the dura mater in the subarachnoid space, which subsequently took on the present fibroid condition.

A noteworthy feature in the case is its chronicity. The fatal termination might have been indefinitely postponed, had it not been for the accident, whereupon an acute meningo-myelitis was lighted up with the accompanying cystitis and bedsores.

One cannot help speculating on the possible good results of an early and vigorous mercurial treatment in this case. Certainly the effects of such treatment are not encouraging in the later stages, as detailed in a somewhat similar case reported lately by Eisenlohr.[1]

Though the neuroglia changes in the posterior and postero-median columns above the lesion are probably of a secondary nature, the same cannot be said of the similar changes in the same regions below. We have here a distinct but early stage of sclerosis. The nerve fibres are diminished in quantity, but their structure is little altered.

At the same time one cannot overlook the well-marked changes in the small arterioles. These are shown in Fig. 4, which is taken from a part of the thickened meninges, and is a rather strongly marked type of the vascular changes in a greater part of the cord. The lumen is much diminished, and

[1] 'Neurolog. Centralblatt,' Feb. 15, 1884; and Abstracts in the present number of 'BRAIN.'

in some there is a distinct proliferation of the nuclei of the intima; beyond this, however, there is in the majority of the vessels no thickening of the intima to the great degree described by Heubner,[1] Drs. Greenfield, Gowers, Barlow and others.[2]

In the dura mater, or rather the most peripheral part of the area of pachymeningitis, we find the vessels not increased in thickness, but the perivascular spaces crowded with leucocytes. Such a condition has often been described;[3] and in an extreme degree by Dr. Buzzard in the last number of 'BRAIN.'

It is tempting to connect the two conditions, and to suppose the hyaline thickening to be a further development of the perivascular infiltration. If this position can be accepted, there is no great difficulty in connecting the fibroid changes in the neuroglia with those of the vessels, when the close communication between the lymph-spaces of the cord and those around the vessels is borne in mind.

Lastly, these vascular changes might be attributed by some to the condition of the kidneys; but I think the fact that they are most affected in the region of the syphilitic lesion is in favour of the theory of an extension of the so-called "gummatous processes" into the perivascular spaces.

[1] 'Die luetische Erkrank. d. Hirnarterien,' 1874.
[2] 'Visceral Syph. Path. Trans.' V., xxviii.
[3] Moxon, 'Lancet,' Sept. 25, 1869. Bruberger, 'Virch. Arch.,' 1874, p. 285.

Critical Digest.

ON RECENT ADVANCES IN THE ANATOMY OF THE NERVOUS SYSTEM.

BY JAMES ANDERSON, M.D.

From the observations of Huschke it appears that the child brings with it into the world one third of the brain-volume it will ultimately possess. It developes the second third in the course of its first year of life, and the remaining third is gained before the twenty-first year. During this first year's rapid development the brain grows 400 ccm., that is more than one 1 ccm. daily, or a piece the size of a bean. Little was known of the histological details of this growth, till Exner showed that of medullated nerve-fibres the infant's convolutions contain practically none. Tuczek and Fuchs now confirm with some modifications the results obtained by Exner, and trace the development of medullated nerve fibres. The former[1] examined by Exner's process four brains; two of children born dead at full time, one of a fœtus of thirty-four weeks, and the other of a female child twenty-seven days old. The parts were placed ten hours after death in ¾ per cent. osmic acid solution, changed in two days, and examined on the sixth or seventh day. Dr. Tuczek summarises his results as follows:—

(1) In all the cerebral convolutions medullated nerve-fibres appear first in the medulla, then in the cortex; and the development of the same extends from centre to periphery.

(2) Medullated nerve-fibres appear earliest—sometimes before the end of the ninth month of fœtal life—in the medulla and cortex of the paracentral lobule, that is, in the median part of the anterior and posterior central (ascending frontal and parietal) convolutions. Next they appear in the occipital lobes and in parts of the Island of Reil. The rest of the cortex contains, in the infant, no medullated nerve-fibres. That is, the law of Flechsig, that medullated nerve-fibres appear first in the region of the pyramidal tracts and *corona radiata*, may be extended to the convolutions connected with them.

(3) The farther development of the medullated nerve-fibres progresses at an equal rate in the convolutions. In the case of the twenty-seven days' child, beyond the paracentral lobule, the central convolutions and the occipital lobe, no convolution contained

[1] 'Neurol. Centralbl.' 1883, No. 20.

medullated nerve-fibres in its cortex. Even in the named convolutions these were contained only in the lower third of the grey cortex. Especially the tangential system of superficial fibres was completely absent.

(4) Medullated nerve-fibres are developed latest in the frontal lobe both on the vertex and the base. The straight convolution, the orbital part of the frontal lobe and the middle frontal convolutions contained in the twenty-seven days' child, even in the medulla, no medullated nerve-fibres.

Fuchs so far confirms the above results[1] that he finds no medullated nerve-fibres in either medulla or cortex of the infant's cerebral convolutions. In the medulla they appear first during the first month of extra-uterine life. In the superficial layer of the cortex they appear first during the fifth month; the second layer (Meynert) shows them first at the end of the first year; the third layer (fibræ arcuatæ) shows them in the seventh month; while the radiating bundles of the deeper layers show them in the second month. After the first year they increase steadily in number and calibre till the eighth year, when they are practically as in adults.

It has for some time been well known that the anatomical description of the spinal cord, as consisting of anterior, lateral and posterior columns of white matter with a double grey crescent in the centre, was a barren topographical division; convenient on certain occasions, as are the "artificial" classifications of plants, but useless as an instrument for scientific progress. We are now learning the same truth in regard to the brain. Dr. Hughlings-Jackson has in his Croomian lectures on the Evolution and Dissolution of the Nervous System, divided the nervous system not anatomically but physiologically into highest, middle and lowest centres, and he has spoken of the evolution of the cerebro-spinal functions from the most automatic to the least automatic, from the most simple to the most complex, from the most organised to the least organised. The anatomical results above detailed are in full agreement with his physiological and clinical generalisations. In such a division of the nervous system as Dr. Jackson has elaborated for its motor side, and in a similar division for the sensory side which, notwithstanding its greater difficulties, will doubtless in the near future be elaborated—we have the "natural" division of the nervous system as opposed to its "artificial" topographical division, and in the recognition of this division we have the promise of a steadier and more scientific advance, both in the physiology and in the pathology of the nervous system. Not that the new physiological subdivision of the nervous system will displace the old topographical. This it will no more do than will Dr. Jackson's classification of the diseases of the nervous system, as dissolutions to various depths of nervous system, displace the clinical classifications at present in vogue. The two divisions, topographical and physiological, will stand side by side, each useful in its own way, each giving useful help to the other.

[1] 'Sitzungsbericht der Wien. Acad. d. Wissensch.,' July, 1883.

At a meeting of the Medico-Chirurgical Society on Jan. 22nd, a very distinct advance was made in our knowledge of the anatomy of the peripheral nervous system by a preliminary communication from Mr. Victor Horsley, in the course of which he demonstrated, by numerous and convincing specimens, the existence of true sensory nerves supplying nerve trunks—of nervi nervorum as opposed to the nervi vasorum, or vaso-motor nerves, already described as supplying the blood-vessels of the epineurium. These nervi nervorum come off at right angles from a primary bundle or cutaneous branch, and terminate, as Mr. Horsley's preparations showed, in end bulbs lying in the perineurium. They have, up to the present time, been concealed in osmic-acid preparations by the fat cells of the epineurium, but can be seen if, as Mr. Horsley suggested, the specimens be taken from an emaciated dropsical subject. This demonstration will afford a safe foundation of fact for any theories as to the cause and treatment of the lightning pains of tabes dorsalis or other neurotic affections.

The 'Brit. Med. Journal' May 24th, 1884, contains an abstract of a Lecture delivered by Professor Hamilton, of Aberdeen, in which he states some of the results of observations made by him, according to the method described in 'BRAIN,' July 1883. The lecture is necessarily given in an extremely fragmentary form, and we defer for the present any extended notice of the points contained in it. That it deals with sufficiently radical and important matter will at once be admitted, when we say that Professor Hamilton, from his observations, believes the corpus callosum to be not a commissure, but the decussation of cortical fibres on their way down to enter the internal and external capsules of the opposite side. The statement practically involves the reconstruction of central cerebral anatomy, and we expect with interest the publication of the lectures *in extenso*.

In the examination of the brain and spinal cord it is frequently difficult to say with certainty whether minute changes observable in the nervous tissue are due to post-mortem changes, to the effects of the hardening reagents, or on the other hand to commencing pathological alterations. The rule therefore with every careful observer is to note, in every case, the period that has elapsed between death and autopsy, the state of the brain and cord on removal, and the processes through which they subsequently pass. Having done this, he describes what he sees, without in doubtful cases deciding as to the exact nature of the minute changes observed. This is not altogether satisfactory, and hence we welcome as extremely valuable the results published by Dr. Richard Schulz[1] of the examination of the spinal cord in twenty subjects of various ages, who had shown no symptoms of spinal disease during life, and whose spinal cords were to the naked eye healthy. The causes of death and all the circumstances of the autopsy are carefully recorded. All the cords were alike hardened in Müller's fluid, frequently changed. They were then placed in gradually stronger and stronger, till finally in absolute alcohol.

[1] 'Neurol. Centralbl.' 1883, Nos. 23, 24.

They were cut with Thomas's microtome in sections $\frac{1}{20}$ to $\frac{1}{40}$ mm. thick, stained with Borax-carmine, partly decolorised by acidulated (HCl) alcohol, and mounted in balsam.

Dr. Schulz discusses in detail the following appearances whose significance is more or less uncertain. (1) *Vacuolation of ganglion cells.*—This consists in the occurrence of two or three clear spaces in the protoplasm of the cells, the nucleus being sometimes visible, sometimes not. It occurred in two out of the twenty cases, and was accompanied by signs of slight imperfection in hardening. Dr. Schulz agrees with Charcot that this is an artificial appearance, and, from the difficulty of hardening the cord in cases of myelitis, he considers that it would be specially liable to occur in myelitis, as also in any condition where the ganglion cells are specially delicate. Leyden considers that vacuolation of the ganglion cells is pathological, and denotes myelitis. Drs. Pick and Rosenbach, who have criticised Dr. Schulz's opinion on this point in subsequent issues of the 'Centralblatt,' also believe that it is pathological, and the latter points out that it occurs in marked degree in the brain and cord of starved animals, and considers therefore that it comes under the head of a degenerative atrophy. (2) *Pigmentation of ganglion cells.*—This consists in a yellowish-brown pigment surrounding, and more or less concealing the nucleus. This was present in a high grade in four cases, aged respectively thirty-two, fifty-two, fifty-three, and seventy-six years; in a slight grade in eleven cases of middle age; while in five young subjects it was completely absent. Dr. Schulz therefore concludes with Charcot that this condition is a matter of age, but much depending on the nutrition of the individual; while it unquestionably occurs also as a pathological change in irritative processes commencing primarily in the neuroglia, also in progressive muscular atrophy and bulbar atrophy. By itself there is no reason to conclude that it is pathological, and Charcot considers that it is the concomitant of the tissue-degeneration and loss of muscular power which accompanies natural old age and the *senium præcox* of Virchow. (3) *Hypertrophy and sclerosis of ganglion cells.*—By hypertrophy of the ganglion cells is meant a swollen, cloudy, opalescent condition of the cell-substance with swollen processes. By sclerosis or atrophic sclerosis of the ganglion cells is meant a shrunken opaque or glancing appearance of the cell substance, with deficient processes. Dr. Schulz in almost all cases found the cells large, with sharp outlines, well-developed processes and distinct nuclei. In seven cases, however, varying from eighteen to seventy-six years of age, the outlines were not sharp, the cell-substance was hyaline in appearance and the nucleus was indistinct. In only two cases were the cells shrunken and hyaline in character. Hypertrophy or hyaline swelling of the cells is therefore not a matter of age, nor of post-mortem change; and while the higher grades are undoubtedly pathological (the result of irritative inflammation, as Charcot, Leyden, and Erb believe), seeing that these seven healthy spinal cords showed the change, it is unwise to pronounce on slight degrees of the change, more especially unaccompanied by other

inflammatory changes. As to sclerosis of the ganglion cells, which usually accompanies only well-marked inflammatory changes, Dr. Schulz considers his cases too few to enable him to pronounce an opinion. (4) *Swelling and hypertrophy of the axis-cylinders.*—The normal diameter of an axis-cylinder, according to Charcot, is ·0033 mm., but in some cases it is as much as ·0099 mm. swollen and irregular in outline. This varicose condition has been observed in the retina, and by Virchow in yellow atrophy of the cortex, and in congenital interstitial encephalitis. All observers agree in regarding it as pathological, occurring in acute and chronic myelitis, in grey degeneration and sclerotic atrophy. Dr. Schulz found the axis-cylinders in eight cords not quite so sharply outlined at the periphery of the cord, this specially where there was slight swelling of the ganglion cells, but nowhere was there an actual enlargement of the axis-cylinder, and the change had quite the appearance of being artificial. Were the swelling of the axis-cylinder a change, artificial or *post-mortem*, Dr. Schulz considers that it would almost certainly have shown itself in some of his cases. He therefore inclines to believe that, when recognised, it may be considered a definite pathological change. (5) *Aggregation of round cells in and about the central canal.*—This Dr. Schulz considers in no way pathological. In some young spinal cords he found the central canal surrounded by a single layer of columnar epithelium; in others there was a growth of round cells around the canal, and in others the canal was completely blocked with round cells. (6) *Neuroglia.*—This in well-hardened specimens is delicate and sharply defined with distinct Deiter's cells. In some cases, however, Dr. Schulz found it not clear, and with no cells visible. This he considers artificial, as the condition was accompanied by swollen ganglion cells and ill-defined axis-cylinders. In six spinal cords, that is in 30 per cent., there were present thin laminæ of fibro-cartilage in the pia mater, and this not in old spinal cords only, but also in those of young adults. It is probably a concomitant of natural or of premature old age.

We have before us an admirable monograph by Dr. William Browning, of Brooklyn, on the veins of the brain and its envelopes. It consists chiefly of anatomical work done by Dr. Browning in Professor Braune's Laboratory in Leipzig, and will undoubtedly in future be the authoritative exposition of this subject. The work contains numerous additions to and corrections of our knowledge of the cerebral veins, one of the principal corrections being as to the course of the Vena Galeni. This it has been the custom to regard as passing directly backward into the straight sinus. A moment's consideration of the relative position of the tentorium cerebelli and the posterior part of the corpus callosum ought to have shown us that such could not possibly be the case, and this is clearly seen in the figure given by Dr. Browning, which shows that the Vena Galeni, passing backward and downward through the median part of the transverse fissure of Bichat, describes a circle upwards round the splenium or posterior part of the corpus callosum, and so at its termination in

the straight sinus "the sinus rectus bears the same relation to the Vena Galeni that a tangent does to its circle." In other words, the Vena Galeni enters *against* the blood-current just as do the superior cerebral veins. Of this somewhat puzzling fact Dr. Browning gives a highly probable explanation in the backward development of the posterior part of the brain carrying its veins with it, and more especially by the late development of the corpus callosum extending backward, and so causing the Vena Galeni, as it were, to circumnavigate it. This explanation, as Dr. Browning states, is corroborated by the more straight course of these veins in the fœtus. Dr. Browning has also carefully examined the important subject of the anastomosis of the cerebral veins. As with the cerebral arteries, so with the cerebral veins, he has nowhere within the brain-substance succeeded in finding a venous anastomosis. For details as to the superficial anastomosis, we must refer to the original work, but we may state shortly that he proves an anastomosis, (1) between the dural and cerebral veins, (2) between the veins of the vertex and those of the base, and (3) between the external veins which join the *portio curvata* of the Vena Galeni and the veins of the base. This last fact shows that "Galen's system of veins is not isolated from the other cerebral veins," but this statement does not apply to the internal veins which run in the choroid plexus and velum. These anastomose in the velum, but nowhere beyond, and thus while obstruction of the straight sinus and of the short common trunk of the Vena magna Galeni can be compensated for, obstruction of both the straight sinus and the compensating basilar vessels, or of the trunks of the Venæ Galeni in the *portio recta*, before they are joined by these basilar veins in the *portio curvata*, means cerebral softening and death.

In the 'Neurol. Centralbl.' 1883, No. 10, Dr. Mendel describes a case of the Ape's Fissure (*Affenspalte*) occurring in the brain of an idiotic girl, eight years of age. Starting in the position of the parieto-occipital fissure just in front of the transverse occipital sulcus, a deep sulcus ran transversely out, ending behind the posterior extremity of the parallel fissure, and thus entirely separating the occipital from the parietal lobe on its convexity. This differs from the cercopithecus and cynocephalus in this region, only in the fact that in these apes the intraparietal fissure and the parallel fissure pass farther back, the first ending in the ape's fissure, neither of which was the case here. That it was really the ape's fissure Dr. Mendel believes was proved by the fact, that on the median surface it communicated with the calcarine fissure in the usual way. Dr. Mendel gives a list of cases where this peculiarity was present, some associated with mental disease, others not. He regards it as an arrest of development. The brain in this case was both smaller and lighter than normal. The frontal convolutions were very small, and the gyrus opercularis round the ascending limb of the fissure of Sylvius was very small, and (owing to absence of the ascending frontal convolution) was in direct continuity with

the ascending parietal convolution. Regarding the question of the homology of the Affenspalte, whether it corresponds with the transverse occipital sulcus or with the parieto-occipital, or with both, as Schwalbe believes, Dr. Mendel has satisfied himself from vertical sections of the brain of cynocephalus sphinx that it corresponds with the parieto-occipital fissure, along with a fissure running forward from it in the depth, termed by Meynert the external occipital sulcus.

For teaching purposes it is frequently important to have the brain of natural size and in a state fit for handling. We make no apology therefore for quoting a process for preserving it given by Germa.[1] (1) The brain is hardened for fourteen days in a saturated solution of bichromate of ammonia. (2) It is washed and placed for one day in a mixture of carbolic acid 1 part, alcohol 1 part, water 8 parts, and glycerine 10 parts. (3) It is washed, and then allowed to dry in the air, and covered with three or four fine coats of varnish; the different parts being coloured, if necessary, with variously coloured metallic powders. (4) Over all is laid on a coat of painter's varnish, for the sake of preservation.

Notwithstanding that the function of speech has now for many years been a favourite subject of physiological and pathological observations, there still remains much to be done before we can be said in any complete sense to understand it. Dr. F. Raymond and M. Artaud[2] have recently made a painstaking and valuable contribution to the subject of the *Intracerebral Course of the Hypoglossal Nerve*. The groundwork of clinical fact and the inferences built upon it are so clearly and logically laid down, that it is difficult in abstract to do justice to their work, which involves a wider range than the actual title of their paper. In writing their paper, the authors had not been acquainted with that by Dr. Ross "On Glosso-labial Paralysis of Cerebral Origin," in 'BRAIN,' July 1882, and their results are therefore the more valuable as agreeing with his.

Affections of speech owing to paralysis of the muscles of articulation have been observed from (1) Cortical lesions; (2) Lesions of the Centrum ovale; (3) Lesions of the internal capsule and crus cerebri; and (4) Lesions of the Pons Varolii. That a lesion of the posterior third of the third left frontal convolution will cause aphasia is now a well-substantiated fact, but that a lesion of the inferior part of the ascending frontal convolution produces glossoplegia or paralysis of the muscles of articulation, is a fact not yet so well substantiated. Of this last the authors cite six cases. In four of these the lesion was located in the inferior part of the ascending frontal convolution, right or left, the result being convulsion or paralysis in the muscles supplied by the hypoglossal of the opposite side from the lesion; and the third frontal not being affected, in no case was the disorder accompanied by aphasia. In two cases both aphasia and glossoplegia was present, the lesions being bilateral and affecting both the inferior and ascending frontal convolutions.

[1] 'L'Encéphale,' 1882, No. 4.

[2] 'Archives de Neurologie,' 1884, Nos. 20 and 21.

From experiment and clinical observation it would seem, according to Charcot and Pitres, that the inferior third of the ascending frontal convolution presides also over the movements of muscles supplied by the inferior branches of the facial, and recently M. Lépine has reported cases of permanent trismus accompanying lesions of this area, or of the subjacent white matter. It forms, therefore, the cortical origin of hypoglossal, inferior facial, and motor root of the trigeminus.

Tracing the hypoglossal fibres downwards, Dr. Raymond and M. Artaud cite seven cases, one recently observed by themselves, of glosso-labial paralysis resulting from injury to the inferior frontal fasciculus of Pitres, arising from the part of the cortex above designated. While the principal symptoms of the progressive bulbar paralysis of Duchenne, viz. paralysis of the tongue, lips and velum palati, were present in these cases, the differential diagnosis is comparatively easy. The symptoms simulating Duchenne's Paralysis are less marked, and differ in their mode of onset. In glosso-labial paralysis of cerebral origin the symptoms commence abruptly, frequently with an apoplectic seizure, accompanied by fall and loss of consciousness; and when the seizure passes off, the patient speaks with difficulty, and cannot move the tongue or approximate the lips. These symptoms are sometimes accompanied by facial paralysis, hemiplegia and intellectual defects, such as are observed in cerebral softening, or by convulsions and epileptiform attacks. In Duchenne's Paralysis the disease is progressive, attacking successively tongue, lips and soft palate, cerebral symptoms being absent; while on the other hand a group of bulbar symptoms are present which are wanting in glosso-labial paralysis of cerebral origin, viz. laryngeal (progressive weakness of voice) pulmonary (attacks of suffocation) and cardiac (syncope). The first disease is also distinguished by the presence of the reflexes, the absence of muscular atrophy, and the conservation of electrical excitability. Death results in the first from fresh cerebral hæmorrhage or softening, or from intercurrent disease of lung or kidney; in the second, from an attack of dyspnœa or syncope.

The fibres of the inferior frontal fasciculus above referred to as arising from the inferior part of the ascending frontal convolutions, pass downward in the internal capsule at the *genu* which divides its anterior from its posterior segment, forming what the authors term the "geniculate" fasciculus, bounded in front by the "intellectual" fasciculus and behind by the pyramidal" or motor fasciculus (from the limbs) and the "sensory" fasciculus. Without being able to render actual proof, Dr. Raymond and M. Artaud believe with a certain probability that the fasciculus of aphasia and that of glosso-labial paralysis pass down in different tracts through the internal capsule and the crus cerebri, the former occupying a position intermediate between the intellectual and geniculate fasciculi. As to the farther course downward of the cerebral fibres of the hypoglossal through the Pons, the authors quote three cases which lead them to believe that they occupy in the Pons the part internal and posterior to the motor pyramids.

Reviews and Notices of Books.

Les Maladies de la Volonté.—Par Th. Ribot. Deuxième Édition. Paris, 1884. 18mo., pp. 177.

The task undertaken by M. Ribot in this volume is to treat disorders of the Will from the same point of view and in the same manner that he has previously found so effectual in the case of disorders of the Memory. Dealing with a subject which is, as the author says, more difficult and less explored than the subject of the sister volume, the success that he attained in his former work is scarcely to be expected in this. Nor would such an expectation be realised. The book affords much pabulum for thought; it frequently compels our adhesion to its doctrines, and it maintains in us a steady conviction that the author is honestly facing his difficulties, which are neither few nor small. But it is by no means so clear, so convincing, nor so free from inconsistency as the author's previous work.

The most interesting and important part of the book is the Introduction, for in this chapter M. Ribot deals with the normal Will, and sets forth his own doctrine concerning both the nature of the state of consciousness that we term willing, and the nature of the physiological process which is the accompaniment, the physical aspect or substratum of the conscious state. This is of course the fundamental problem upon which the whole of the rest of the book depends; and when the method of its solution is known, the mode of treating the portion of the subject indicated by the title can be to a considerable extent forecasted.

After mentioning the problem of free-will, merely to place it on one side as beyond and apart from the scope of his enquiry, M. Ribot postulates that dualism of which he is a steadfast champion, and states as the foundation of his doctrine the elementary principle that every state of consciousness has always a tendency to express itself—to issue in a movement, in an act. This principle is but a particular case of the general law that all nervous action is reflex. The most refined and original exercise of Will is but an immensely developed reflex action, accompanied by a peculiar state of consciousness. The continuous gradation that unites these extremes is divided by M. Ribot into three stages, the division being understood to be arbitrary. First, there are those mental states which issue in action with a certainty and rapidity almost equal to reflex action. Such are the passions and the "fixed ideas." Second, come those cases to which the term Will is usually

applied—cases in which a resolution issues in an act after more or less deliberation; and the third group consists of abstract ideas, whose tendency to issue in action is very slight. Each more elaborate mental state is less apt to issue in action than the less elaborate states, because, in the first place, its anatomical substratum includes fewer and feebler motor elements; and in the second, because the substratum is, as a whole, less directly connected with the motor tract.

Will does not, however, manifest itself solely in movement. It is also displayed, and in its most striking and characteristic instances, in arrest of movement. That the voluntary arrest of movement is a higher development of the physiological process of inhibition, is sufficiently obvious, but as to the intimate nature of either process M. Ribot does not add much to our present knowledge. He takes, as is usual, the influence of the vagus on the heart as the simplest example of inhibition; but in the light of recent researches it becomes extremely doubtful whether this is so, and whether the inhibition of the heart's beat is not indeed an extremely complex and altogether exceptional instance of this process. The current notion of inhibition scarcely gives to it a position or a scope commensurate with its importance and its prevalence throughout the nervous system; although the doctrine of "loss of control," which implies the doctrine of universal inhibition, has long been domiciliated amongst us. If there is such a thing as excessive action of a nerve-centre, permitted by the removal of the function of the centre above, then it is only saying the same thing in another way to say that the superior centre normally inhibits the inferior centre. And just as we have already so modified our conception of co-ordination as to ascribe it, not as a special and separate faculty to a single centre or region of the nervous system, but as a universal attribute to every centre in every region, so we must extend our conception of inhibition. It is as absurd to speak of some centres as motor and others as inhibitory, as to speak of some centres as motor and others as co-ordinating. We must recognise that the power of inhibition is as integral and necessary a part of the function of every nerve-centre as the process of discharge or the attribute of co-ordinating. What is the meaning of the increase of reflex action in the lower section of a divided spinal cord, but that the inhibition of the nerve-mass above is removed from it? What is the meaning of the rigidity of a hemiplegic hand, but that the inhibition of the cerebral centres is removed which counteracted the nervous afflux that maintains the rigidity? From top to bottom of the hierarchy of the nervous system every centre inhibits those below, and is inhibited by those above. On any other supposition the hypothesis of over-action from loss of control is nonsense. The inhibition exercised by the highest of all centres upon the whole mass of strata below it, is the physiological side of the voluntary arrest of movement, just as the motor discharge of those highest centres is the physiological process in voluntary action. What the precise molecular mechanism may be by virtue of which the same centre can act on

its subordinates, now as an inhibitory or restraining, and now as a permissive or exciting influence, we are at present ignorant; but unless we are to recast the whole of our beliefs as to the mode of nervous action, both physiological and pathological, we must accept the doctrine that each centre has this multiplicity and contrariety of function.

M. Ribot seeks the explanation of voluntary arrest in other quarters. He finds first that certain mental states—fear and pain for instance—have a primarily depressing effect. That is to say, that the occurrence of these feelings is always and of necessity accompanied by a diminution, or even an abolition for the time being, of voluntary action. Then he goes on to show that in the process of education voluntary movements are counteracted and suppressed by inducing pain or fear in the pupil—by threats and reproofs. By the law of association, the recurrence of the desire to perform the forbidden act will of itself arouse the memory of the reproofs and the threats, will induce a repetition of the accompanying state of depression, and hence will provide its own antidote. Doubtless the hypothesis is correct, so far as it attributes the suppression of the movements to the preponderance of counteracting motives aroused by the law of association; but that this counter-influence is a recurrence of the depression, and that it is the general depression of nerve discharge that prevents the occurrence of the movement, is a supposition that does not appear to be borne out by facts. Such an hypothesis would reduce the restraining influence of duty and honour to the level of mere paralysing agencies, a conclusion that would be not only ethically disheartening, but opposed to the daily experience of all men. If the stern check of duty is in the last resort a mere depression of heart and brain action, rendering us powerless to act wrongly, whence the feeling of strife, of effort, of strenuous conflict that accompanies the resolve not to do this forbidden thing? No; on the strictest adherence to evolutionary doctrine, as well as on grounds of common experience, the acquisition of the power of voluntarily inhibiting an act or a set of acts must be looked on as expressing the formation of a new stratum in the nervous centres, the addition of a new and superior rank to their hierarchy, the superposition of a new plexus, which has by virtue of its position and connections the power of inhibiting, as well as of arousing, the action of all that are subordinate to it.

When M. Ribot speaks of the will as the reaction of the entire individual upon the entire group of circumstances that affect him, all who have studied the subject will probably agree with him, and when we remember that as we ascend the hierarchy of nervous centres, each rank that we reach represents a larger and larger portion of the organism, until the highest centres of all represent the whole organism most completely, it is easy to see that from a physiological point of view the action of these highest centres is equivalent to the reaction of the entire organism.

Having laid his foundation in the description of the normal Will, M. Ribot applies the principles therein educed to the consideration

of the disorders to which volition is liable. While it is not possible, nor, in so complicated, abstruse and difficult a subject, is it to be expected, that his doctrines will always meet with the reader's assent, yet every one of them is felt to be worthy of careful consideration, many of them are novel and ingenious, and all are founded on the firm base of the principle of evolution. The author's method is thoroughly scientific, and his opinions, if they do not always command our assent, invariably compel a respectful and attentive consideration. CHARLES MERCIER.

Etude Critique et Clinique de la doctrine des Localisations Motrices dans l'écorce des hémisphères cérébraux de l'homme. Par M.M. les Profs. CHARCOT et PITRES. (Reprinted from the 'Revue de Médecine'). 1 vol. 8mo., pp. 120. Paris: Alcan, 1883.

THE authors in a series of articles originally contributed to the 'Revue de Médecine' have furnished an interesting and valuable research on the subject of cerebral localisation. The basis of this inquiry is an analysis of one hundred and eighty-five cases of brain disease which have been carefully reported during life, in comparatively recent times, and in which opportunities were given of demonstrating the pathological condition after death. The general result of these observations is that there is no well-authenticated instance of a destructive lesion outside the cortical motor centres causing permanent paralysis; and on the other hand, that there is no rigorous observation of a destructive lesion of this motor zone which has failed to produce paralysis on the opposite side of the body. In short, they maintain that the facts opposed to the doctrine of central motor localisation have not been established.

The conclusions arrived at as a result of the entire enquiry are as follows:—

1. The cortex of the cerebral hemispheres in man may be divided, functionally, into two parts: motor and non-motor, according as destructive lesions do or do not cause permanent paralysis of the opposite side of the body.

2. The *non-motor zone* comprehends: *a*. All the pre-frontal region of the brain (orbital lobe, 1st, 2nd, and 3rd frontal convolutions). *b*. All the occipito-parietal region (occipital lobe, superior and inferior parietal lobules). *c*. All the temporo-sphenoidal lobe.

3. The *motor zone* includes only the ascending frontal and ascending parietal convolutions and the paracentral lobule.

4. The paralyses produced by destructive lesions of the motor zone have different clinical forms according to the seat and extent of the lesion. Thus, complete hemiplegias of cortical origin are produced by diffused lesions of the ascending convolutions; partial

paralyses are produced by circumscribed lesions of these same convolutions. The localisation of the lesion in some of these partial paralyses or monoplegias can be determined. MM. Charcot and Pitres announce the following as data for such determination: *a*. The brachio-facial monoplegias coincide with lesions in the inferior half of the ascending convolutions. *b*. The brachio-crural monoplegias coincide with lesions in the upper half of the ascending convolutions. *c*. The facial and lingual monoplegias depend upon very circumscribed lesions of the lower extremity of the ascending convolutions, particularly of the ascending frontal. *d*. The brachial monoplegias depend upon a very limited lesion of the motor zone: particularly of the middle third of the ascending frontal. *e*. The crural monoplegias depend upon very limited lesions of the paracentral lobule.

5. Whether those paralyses caused by destructive lesions be general or limited, they will in time be followed by secondary contractures of the paralysed muscles, with descending degenerations of the voluntary pyramidal tracts.

6. Irritative lesions of the cortex can give rise to epileptiform convolutions (partial or Jacksonian epilepsy), which are different from those of true epilepsy. They begin with a motor aura, and may be either generalised, or remain limited to one side of the body or one group of muscles.

7. In general the irritative lesions which cause epileptiform convulsions are localised in or near those centres which, if destroyed, would cause paralysis in the muscles affected by the convulsion. But these lesions may be even in the non-motor zone, and their relation to the functionally affected centres is not so close as is the case with paralyses and destructive lesions.

The study of the cases of monoplegia of the face, arm, or leg is particularly instructive. The authors give several figures representing the accurately circumscribed lesions which give rise to these localised paralyses. In presence of the accumulated proofs of clinical observation on the one hand, and of pathological and developmental anatomy on the other, all pointing to the same conclusions, viz., to the separation of the sensory from the motor spheres, and to the independence of the several motor centres in the human brain, the physician may look with composure upon the contradictory results of the physiological laboratory. It is quite admissible to believe that the functions of an organ of such gradual evolution in the animal series as the cerebral cortex may, according to the law of differentiation, be far more strictly localised in man than in the lower animals; and in such a wider synthesis, perhaps, the results of experimentation and clinical observation will be found to be reconcileable, nay to support one another, and we may thus hope to learn not only how things are, but also how they came to be.

A. H. Bennett, M.D.

A Statistical Inquiry into the Nature and Treatment of Epilepsy. By A. Hughes Bennett, M.D.—H. K. Lewis, London, 1884.

This little book consists of three papers, which have already appeared in the Medical Journals at different dates during the past few years. They are now republished together, so as to form a connected inquiry. They constitute a careful and elaborate statistical investigation of many points in the clinical history of Epilepsy, and more especially of the effects of the bromides in the treatment of that disease. The author in his researches is actuated by the opinion, that the science and art of medicine is to be advanced by the collection of well-recorded facts, rather than by statements and unsupported assertions; and however laborious this may be to the physician and unattractive to the student, he believes that it is the only means by which sound therapeutic truths are to be arrived at.

The first chapter deals with a statistical investigation into the Etiology and Symptomatology of Epilepsy, and the percentage occurrences of many points connected with these conditions are given in detail. The relative frequency of the various predisposing and exciting causes in a given number of cases, as well as many of the chief symptoms and their phenomena, are demonstrated in tabular form, in such a way as to be easily accessible for reference.

The second chapter is an important contribution towards the exact estimation of the effects of the bromides on epileptic attacks. Here an attempt is made to determine, not the curative properties of the remedy on the disease itself, but only its actions on the chief symptoms, the paroxysmal seizures. This is carried out in a most systematic manner after the following plan, which shows very clearly the results of the remedy, as far as statistics are capable of demonstrating so complex a subject. From a long series of unselected cases only those were utilised for the present inquiry who had been continuously under treatment for a minimum period of six months, to a maximum of four years. In such instances all the facts of the case were carefully recorded, and more especially the average monthly number of attacks prior to treatment was determined. The drugs were then administered and steadily persevered in, and in the same way the number of attacks noted. In this manner the particulars of 117 cases have been collected and arranged in tabular form, the accumulating and recording of which represents a great amount of patience and labour. Further investigations were instituted to ascertain whether the effects of the bromides were influenced by any special peculiarities connected with the disease, such as its form, its period of occurrence, its hereditary nature, the age at which it appears, the chronicity of the disorder, the state of health of the sufferer, and

so on. In all these conditions special researches have been made, and tables drawn up to indicate the results of the treatment in each. The general results arrived at are as follows: 1. In 12·1 per cent. of epileptics the attacks were completely arrested during the whole period of treatment by the bromides. 2. In 83·3 per cent. the attacks were greatly diminished, both in number and severity. 3. In 2·3 per cent. the treatment had no apparent effect. 4. In 2·3 per cent. the number of attacks was augmented during the period of treatment. 5. The form of the disease, whether it was inherited or not, whether complicated or not, recent or chronic, in the young or in the old, in healthy or diseased persons,—appeared in no way to influence treatment, the success being nearly in the same ratio under all these conditions.

The third chapter attempts to investigate the effects of the prolonged administration of the bromides on the epileptic patient, with the view more especially of ascertaining, whether in modifying the number of attacks, they in any way injuriously influence the health of the patient. The data upon which conclusions are founded consist of 141 cases, continuously under the influence of the bromides for a minimum period of twelve months, and extending to a maximum of six years. The dose of the drug was from one and a half drachms daily, upwards. These observations are also the result of an arduous and prolonged experience. The general results obtained are that, while the bromides arrest or modify the attacks, as already indicated, they, contrary to the usual belief, do not in the majority of cases seem injuriously to affect the mental or physical well-being of the patient. Tables are drawn up to show the approximative results, more especially on the physical and mental powers, on the general nervous system, and on the production of cutaneous affections. The details are given of four interesting cases, who for no less than six years have regularly consumed daily one and a half drachms of the bromide. In all of these the patients were in good physical and mental condition, and able to earn their livings by their respective occupations. The facts further seem to indicate that the symptoms of bromism diminish in intensity, the longer the remedy is persisted in. They also seem to suggest that the epileptic is more tolerant of the drug than an ordinary individual not suffering from that disease. As to whether the treatment finally eradicates the disease, there is no positive evidence to show, but all the facts lead to the conclusion, that if it was systematically carried out for a sufficiently long period, this is not impossible.

Finally, the author throughout recognises the extreme difficulty of interpreting facts—estimating results, as well as the many possible fallacies that beset the path of the investigator. While the conclusions, for the sake of convenience, have been arranged in tabular form, it is admitted that these are only approximative, and cannot be calculated with mathematical precision. At the same time the details of the inquiry have been collected with such care, and recorded with such accuracy, that it is believed they represent in the main the true facts of the case. We

therefore consider that the present work of Dr. Bennett constitutes a substantial contribution to what is a very important subject, and hope his example will find numerous imitators; for nothing tends to cast discredit upon therapeutics so much as premature conclusions from observations both too few in number and too superficially performed, and the hasty publication of isolated cases alleged to have been "cured" by some drug or other.

A. DE W.

"*Text-book of the Principles of Physics.*" By ALFRED DANIELL, M.A., Lecturer on Physics in the School of Medicine, Edinburgh. (1 vol. 8vo, pp. 653. London: Macmillan & Co.)

THE task of physiology and pathology is double. First we must define and localise the function, or symptom (used in the sense of disturbed function). Second, and this is the truly scientific aspect of the problem, we must explain the function, and reduce it to its component factors. These factors are of a physical and of a chemical order; they are forces at work under conditions, the complexity of which grows with the evolution of the tissues of which they are properties. Whether there is a special force or a special modification of the physico-chemical forces, a vital force, directing the developmental and evolutionary *nisus* of organisms under varying conditions and surroundings, is a question still unanswered, at least for those whose minds are not fully satisfied by current theories. But whatever view be the correct one, physiology and pathology have essentially to do with force; the description of the mechanisms upon which the manifestations of force, the storage, liberation and transformations of energy, depend, is a mere subsidiary operation preliminary to the discovery of the laws which govern the phenomena of force manifested as life.

Now the method of studying nature is to proceed from the simple to the complex; and as in mathematics it would be idle for one to attempt to solve the problem of integral calculus who had not previously mastered the principles of arithmetic and simple algebra, so it is hopeless for one ignorant of those conceptions of force which form the basis of physics and chemistry, to launch into disquisitions concerning nerve-force and the other manifestations of energy in living organisms. Elementary as this principle seems, yet our medical literature affords ample proofs that it is very generally discarded. There is a natural tendency in the human mind to lend objectivity to its concepts; it is the most ignorant who are the most prone to give explanations because they are unconscious of the ridiculous inadequacy of their data for the solution of problems, the depth of which they cannot even distinctly appreciate.

The study of modern physics forms an essential propædeutic

to that of biology. Treatises on the subject have ceased to be mere collections of experimental facts, regarding heat, light, electricity, &c., without apparent connection between them; their object is, as Mr. Daniell says, to teach the whole of Natural Philosophy as substantially a single science, in which scattered facts are connected and co-ordinated by reference to the principles of dynamics and the great experimental law of the conservation of energy. The distinction, too, between physics and chemistry is becoming more and more clearly one of convenience only; the phenomena with which they deal are but instances or embodiments of intimately correlated ideas, and it is in a study of this synthesis of the laws governing the simpler properties of matter that they shall find a most useful discipline who desire to penetrate more deeply into the mysteries of living organisms.

The book now before us is of considerable value from a double point of view. First, it is a very able one, and which fully fulfils the intention of its author to present a connected account of the principles of physics suitable to the preliminary training of medical men. Secondly, it is the only book of its kind in the English language; for though we have numerous treatises of the highest merit on the subject of Physics, yet they are all monographs. I do not mention the well-known books of Deschanel and Ganot, for they are translations only of works originally written on the now antiquated type of manuals of physics; and though their able English editors have spared no pains to add largely to their scientific value by supplying much new matter, these manuals lack the continuity of thought which is the distinctive characteristic of Mr. Daniell's book.

We need not enter here into any details as to the actual contents of the volume before us, which, we are glad to see, has been exceptionally well received by physicists generally. The author's position as Lecturer in a Medical School has enabled him to produce a work well adapted to the wants of those who wish to base their physiological and medical studies upon more solid foundations than those offered by the very imperfect curriculum still in vogue at many of our Schools.

A. DE W.

L'âme est la fonction du Cerveau. By EMILE FERRIÈRE. 2 vols. small 8vo, pp. 436 and 404. Paris, Germer Baillière, 1883.

THIS work consists mainly of an account of the nervous system as the physical substratum of the psychical phenomena of man and of animals; and of the various manifestations of mind under varying conditions of brain-development and brain-disturbance. It is a curious fact that, whilst the author starts with the professed intention to show the consonance of Spinoza's theories on mind or matter with the data of modern anatomy, physiology, and

physiological psychology, his efforts should have resulted in producing a generally accurate and very readable summary of our present knowledge on the subject. The reference to Spinozism in the preface would lead one to expect some excursions at least in the field of the metaphysics of monism; but such is not the case. The author remains throughout a positivist indeed, never rising above the purest phenomenism. He shows the parallelism between nervous organisation and mental manifestations, and contents himself with calling the latter a *function* of the former. It would have been well if he had defined this word "function," which to us seems to explain nothing, and leave the problem—an insoluble one according to most competent thinkers—very much where it was.

When we said that the metaphysical aspect of the question has been left untouched—nay, apparently ignored—by the author, we only referred to the constructive part of his book. There runs throughout its pages a vein of controversial aggressiveness against the popular "Spiritualistic" conception (still represented in the official philosophic teaching in French Academies) of a dual nature of man, who according to this theory consists of an independent spiritual entity united to a material organism, of a spirit and a body mysteriously acting and reacting upon one another. It is possible that for the general reader this constant repetition that the psychical phenomena observed in man and the lower animals, in health and disease, are not compatible with this view, may be found advantageous or necessary; but to the student of either of the great metaphysical systems or of modern psychophysiology such an instance will appear somewhat puerile. In the course of his controversy M. Ferrière lays considerable stress upon an argument by analogy, which however appears to us more specious than solid. He tries to show by a parallel between the attributes of mind as a function of the brain, and those of magnetism as a "function" (*sic*) of the magnet, that there is less difference between these "immaterial forces" than between the material products of two glands, such as the spermatic and lactic secretions. True, magnetism disappears when the outer film of the magnet is dissolved in an acid, just as psychical manifestations do when the cortical layer of a brain is removed; but how superficial are all such analogies! Magnetism is a force correlated to heat, light and electricity, and postulates space for its manifestations; mind is subject to conditions of time only, is not a force, and does not originate in any transformation of the forces set free by that liberation of energy which constitutes nerve-discharge. Nor can mind, not being a force, ever liberate energy, and originate any of the cerebral processes which are the necessary conditions of thought, feeling, as well as of movement; it is neither a cause nor an effect of the physical changes involved in nerve action; it neither precedes nor follows, but accompanies those changes. Mind is coextensive with consciousness; if it were the product of brain action, the equivalent of a certain quantity of nervous energy which might be supposed to be converted into vibrations (of a mentiferous ether, as it has

been suggested) our mental processes would be uniformly proportional to the quantity of nervous matter oxidised: but this is not the case, and identical cerebral acts differ largely as to the intensity of the conscious states which accompany them. In other words "cerebration" and "mentation" do not both obey laws reducible to those of transformation of energy. There is no common term between the reflex process in which a nerve excitation from the sensory sphere liberates in a motor centre a certain quantity of stored-up force, which in its turn sets free energy in certain groups of muscles, and the mental state we call a volition.

M. Ferrière, like many students of neurophysiology, has been so dazzled with the apparent simplicity of the method, now so popular, of expressing psychical in terms of physical phenomena—mind in terms of matter—that he has lost sight of the very problem which he gave himself the task to solve. The fact is that we must either remain content to leave the answer in the domain of the unknowable, or launch into the abyss of metaphysics. Mind, in Spinoza's own language, is the idea, or, as we should now call it, the consciousness, of the body; not a "substance" but a "mode"—a collection of states. Body is the subject of the soul; both are modifications of one substance with attributes infinite in number, but cognisable to us under two only, matter and thought. Hence mind and body are but one phenomenon under two aspects—the same ray of divine light, refracted as it were, and analysed through the prism of psychological introspection. Even from the standpoint of our modern positive science many will feel more sympathy for the bold speculations of the great metaphysician, than for the so-called materialistic solution of the problem which consists in suppressing one of its terms.

This book is very readable, and full of information on every topic connected with the nervous system considered as the physical basis of mental manifestations. The style is very clear and the facts well arranged. The chapters on the anatomy and embryogeny of the nerve centres, however, might have been advantageously either curtailed or illustrated.

A. DE W.

Neurypnologie; Traité du sommeil nerveux, ou hypnotisme. By JAMES BRAID. Translated into French by Dr. J. SIMON; with a preface by Prof. BROWN-SÉQUARD. 1 vol. small 8vo, pp. 268. Paris: Delahaye, 1883.

THE study of the phenomena of somnambulism, or hypnotism, has during the last few years been prosecuted with much energy and success, and we are glad to find the suggestive work of Braid placed before continental readers in the shape of an excellent French translation. This volume will be welcome in England also, both on account of the scarcity of Braid's original edition

(1834), and of the new additional chapter which it contains. This chapter consists of a communication made by Braid in 1860 to the Academy of Sciences, in the very month of his death, and occupies thirty-five pages in the translation.

The "Neurypnology" is divided into two parts. The first, historical, experimental and controversial, contains an *exposé* of Braid's methods and theory. His merit consists in having exploded the "animal magnetism" craze, and shown that the hypnotic condition is induced by sensorial impression of various descriptions. Instead of the classical "passes" made over the patient by the operator's hands, he used the convergence of the eyes inwards and upwards, such as in fixation of a small object placed near and above the place of ordinary vision.

This portion of Braid's work is too well known for us to say any more about it here. The second part is therapeutical, and though at the time of publication it aroused a considerable amount of ill-feeling against the author, it has now passed into comparative oblivion. But the time is near when the curative influence of hypnotism will be submitted to the same scrutiny as its physiological and psychological import has undergone. There is certainly much exaggeration and self-deception in Braid's enthusiastic report of the cases he declares to have been successfully treated by himself. Still there is no doubt a substratum of truth in what he says; and we have no right to refuse *à priori* to believe an observer of such undoubted uprightness when he begins to speak of the practical applications of a discovery in the elaboration of which he has shown true scientific insight.

The cases in which hypnotism appears to have relieved the symptoms more or less permanently belong chiefly to the group of neurotic functional disturbance; the method consists simply in hypnotising the patient (several times if necessary) and allowing him to recover naturally from the somnambulic state. A few well-observed instances in which this has been efficacious have been quite recently published in Germany, where Profs. Preyer and Heidenhain's physiological researches on hypnotism have excited a considerable interest. Thus Dr. Wiehe ('Berliner Klin. Wochenschrift,' January 1884) experimented upon four patients in Prof. Bäumler's Clinique at Freiburg. Two cases (of hysterical nature), in which persisting and violent clonic spasms formed the chief symptoms, were the one relieved, the other cured of the complaint. A third patient was freed of hemi-anæsthesia; and the fourth, who had been vainly submitted to electrical and other treatment for neuralgic pains in the face and arm, was completely relieved of the symptoms by one hypnotisation. In other cases of hysterical disturbance, however, hypnotism failed. It is not to be forgotten in experiments of this kind that a repetition of the operation may prove injurious to the nervous system—for instance, leave a tendency to spontaneous somnambulism in predisposed subjects. Hypnotism may in certain cases prove a valuable substitute for the usual method of inducing anæsthesia; Braid and others have reported instances where surgical operations have been carried

out painlessly on hypnotised subjects. But whatever be the practical value of hypnotism from a medical point of view, the light it has already thrown upon the mechanism of cerebral action, and the important place it must henceforth occupy in experimental psychology, are enough to encourage fresh efforts in the investigation of its phenomena. The publication of the "Neurypnology" is a well-timed homage to the memory of a man who, with singular tenacity of purpose and true singleness of heart, incurred much obloquy and persecution in the pursuit of science and truth.

A. DE W.

Medicinische Bibliographie. By DR. ARTHUR WURZBURG, Librarian to the Imperial Sanitary Office in Berlin. Vol. I. 1883, 8vo, pp. 386. Leipzig: Breitkopf and Härtel.

THIS publication will be found extremely convenient by everyone who wishes to be kept *au courant* of the ever-increasing literature of the medical sciences. For the modest sum of six shillings the subscriber receives weekly a part of six to eight pages, in which all the recent works and papers of importance are indicated by title and reference. They are arranged under about twenty-five headings, one of which, that of nervous diseases and psychiatry, will prove of special interest to our reader, and keep him informed of the latest papers to which he may have to refer.

The size of this 'Bibliographie' excludes, of course, the possibility of its pretending to the same exhaustive completeness as the American 'Index Medicus,' now defunct, we fear, for want of support by the profession at large. It will nevertheless be well received, as it thoroughly fulfils the eminently practical design of its able Editor.

A. DE W.

Clinical Figures. Brain: I. after ECKER; II. after PITRES. Lebon & Co., 28, Southampton Buildings, Chancery Lane, London.

THESE figures are published on sheets of paper, gummed at the back, so as to be readily transferred into a note-book. Each sheet contains six figures, and there are twenty-five sheets in a book. The first series comprises views of the surface of hemispheres (superior and inferior, right and left external, right and left internal aspects); the second reproduces the very useful sections of Pitres. This publication fills a long-felt want. They will be found invaluable in the post-mortem room; also exceedingly convenient to the student of cerebral pathology, who is thus enabled to take rapid sketches of the interesting localisations of brain-disease with which he meets in the course of his reading.

Messrs. Lebon have conferred a further boon upon neurologists by announcing that they will supply the electroplate-block of any of the twelve figures, with the outline of the lesions which it is desired to illustrate. All that the author has to do is to indicate on one of the diagrams the position of the hæmorrhage, tumour, &c. His markings are transferred upon the cliché, the printed figure from which shows the lesion as a lighter patch upon the darker ground. The cost of illustrating cases of brain disease is thus reduced to a very trifling sum (about two shillings for each figure).

Messrs. Lebon are preparing a similar series of diagrams of the spinal cord. Other series have been published, illustrating the eye and retina, the surface of the head, of the body, &c., all of which will occasionally be found useful for a graphical record of the symptoms observed in cases of nervous disease.

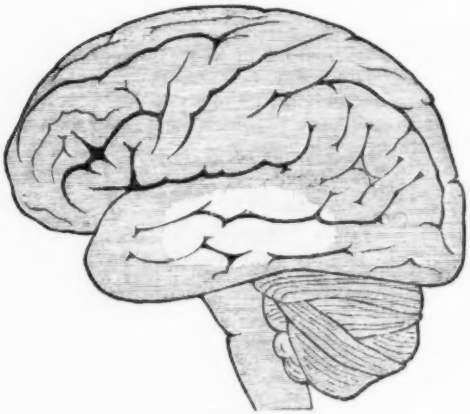


FIG. 1.

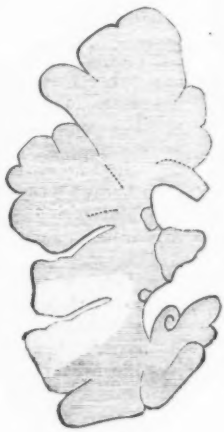


FIG. 2.

As specimens of the clinical figures of the brain, and to show the way in which localised lesions may be illustrated by their means, I have appended two figures illustrating a patch of softening in the left temporal lobe. Figure 1 depicts the superficial extent of the same; Fig. 2, its depth on a pediculo-parietal section of the brain. The case from which they are taken is one of *verbal deafness* recently published by Prof. Tamburini, and which will be referred to more fully in a future number of 'BRAIN.'

A. DE W.

I. J. ROSENTHAL *and* M. BERNHARDT: *Electricitätslehre für Mediciner und Electrotherapie.* Berlin: Hirschwald, 1883. 521 pp.

II. S. TH. STEIN: *Die Allgemeine Electrisation des menschlichen Körpers.* Zweite Auflage. Halle: Wilhelm Knapp, 1883. 144 pp.

I. THE first part of this work (12 chapters) is the third edition of Rosenthal's well-known book on Medical Electricity, carefully revised and adapted to the present standard of this discipline. For English students it is perhaps a little too thorough, in so far as the author has not limited himself to those facts which the practitioner requires to be acquainted with before he can make a practical use of electro-therapeutics, but he also gives an account of facts and methods which are used in physiological research. This, however, can hardly be considered as a deficiency of the book, for the author treats the subject with much conciseness and remarkable perspicuity of style.

The second part is Professor Bernhardt's own work. It contains 16 chapters on the application of Electricity to the diagnosis and treatment of diseases. Two chapters are devoted to remarks on batteries, galvanometers and other apparatus, and to an account of the motor points and the methods of investigations of the motor nerves and muscles. Then follows a description of the pathological alterations of electric irritability, and of the therapeutical effects of Electricity in general. The greater share of this part of the book belongs to the description of the therapeutical application of Electricity. An Appendix on Metalloscopy, Metallotherapy, and static Electricity, and a number of interesting clinical observations, chiefly illustrating the use of electro-diagnosis, completes the book.

It is hardly necessary to say that Professor Bernhardt, whose works represent an essential part of electro-therapeutical literature, has fulfilled his task in a masterly style. The book is free from merely hypothetical reasoning; it deserves to be carefully studied by all those who take an interest in the diagnosis and treatment of diseases of the nervous system, for which purpose a thorough knowledge of the medical applications of Electricity is absolutely indispensable.

II. The fact that the first edition of this little book has been sold out within six months is a striking proof of its practical value. The chief object of it is to show the use of Electricity applied to the entire surface of the human body (or the greater part of it), especially in cases of neurasthenia and similar affections. This kind of treatment was first recommended by Beard and Rockwell, of New York; but while the American authors exclusively applied the faradic and galvanic currents, Dr. Stein also advocates the use

of static electricity or Franklinisation. He refers to Charcot's and Vigouroux's experience, to which he adds a number of observations of his own. The electric bath, too, is extensively described and much recommended. Besides this, the little book contains many useful hints concerning the choice and handling of electric apparatus; it is in every way an interesting publication, even for those readers who may not be able entirely to agree with the author's views and expectations.

R. H. Pierson, M.D. (Dresden).

Hughlings-Jackson on Evolution and Dissolution of the Nervous System.—(*Croomian Lectures for* 1884. *Brit. Med. Journal*, March 29th, *ff.*)—The Croomian Lectures were delivered in March of this year at the Royal College of Physicians by Dr. Hughlings-Jackson, who chose for his subject the "Evolution and Dissolution of the Nervous System," and gave what may be regarded as a highly condensed summary of the results of the work which has occupied him for many years. At frequent intervals Dr. Jackson has published, in somewhat fragmentary form, the results that he has arrived at in localised districts of his field of work; but these publications have been distributed over so long a period, and disseminated in so many journals, that they are not readily available for reference. These lectures are therefore welcome as a recapitulation in an accessible form of much that has appeared before; as giving the latest outcome of Dr. Jackson's labours; and as presenting his views in a more unified, or as he himself might term it, a more integrated shape than they have yet assumed. Dr. Jackson regards the central nervous system as a hierarchy; in which each grade controls the grade below and is controlled by the grade above; each grade represents over again and co-ordinates in more elaborate combinations the parts represented and co-ordinated by the grade below; and every part or region or centre of a grade represents a larger share of the organism than any corresponding part of the grade below, a more limited share than any corresponding part of the grade above. Thus each centre in the lowest grade represents but a limited portion of the organism, and each centre in the highest grade represents the whole of the organism, but in no two of the latter centres is the whole organism represented in quite the same way. The whole of the grades are grouped in three main divisions —lowest, middle, and highest centres. Of these the lowest are the most completely organised, the highest the least completely organised. When disease attacks the nervous system, there are always two sets of manifestations: negative and positive—loss of function and excess of function. The functions lost are those of the centres diseased. The functions that are excessive are those of centres subordinate to the centres diseased, and permitted to act excessively

by the removal of the control normally exercised by the centres now diseased. The centre whose function is abolished may belong to the lowest, to the middle, or to the highest division, affording examples of local dissolution. On the other hand there may be a uniform dissolution, the whole of the highest grade of all being first lost, and successive grades being pared off, as it were, as if in layers. Dr. Jackson applies these doctrines, with many subsidiary hypotheses, to every kind of nervous disorder, from atrophy of muscles to insanity, and from giddiness to coma; and from his vast knowledge of diseases of the nervous system he is able to supply an apparently limitless profusion of instances to every section and subsection of his subject. C. MERCIER.

Ballet on Exophthalmic Goitre. (*Revue de Médecine*, April 1883.)—The general results at which the author arrives after an inquiry into this subject are as follows:—

1. To the classical symptoms of exophthalmic goitre (palpitations, swelling of the neck, tremor,) there are occasionally added others which, like them, are attributable to disorders of the nervous system.

2. These symptoms are on the one hand *convulsive* (epileptic or epileptiform attacks), or on the other *paralytic* (hemiplegia or paraplegia); also pretty frequently albuminuria, glycosuria, or simple polyuria may be observed.

3. These convulsive or paralytic complications appear to arise, not as the direct result of the Basedow's disease, but of another coincident neurosis (epilepsy, hysteria).

4. Sometimes certain convulsive phenomena (epileptiform attacks) seem intimately associated with exophthalmic goitre itself, and the special clinical conditions under which they are manifested authorise us in connecting them with disturbance of the cerebro-bulbar circulation, itself occasioned by perturbation of the action of the heart.

5. Among the paralytic disturbances some are of minor importance such as the weakness of the hands, the temporary paresis of one or both upper extremities, and the feebleness of the lower limbs. They may be looked upon as directly dependent either upon the tremor, or on functional derangement of the cerebral circulation.

6. The polyuria, albuminuria, and glycosuria are probably more frequent than might be supposed from previous researches, and they indicate a derangement of the bulbar innervation.

A. H. BENNETT.

Dr. Franz Zimmerlin on Hereditary (family) progressive muscular Atrophy. (*Zeitschr. für Klin. Med.* Band 7, Heft 1.) —The clinical picture of hereditary ataxy has been classically worked out by Friedreich; the hereditary form of progressive muscular atrophy by Leyden. The hereditary form of progressive muscular atrophy, in opposition to the not hereditary, is classified as a clinical secondary form by Leyden, as follows:—

Beginning of illness nearly always in advanced childhood, seldom later, attacks several members of same family, particularly males. Begins with weakness and atrophy in loins and lower extremities, first in lower part of leg or muscles of back; it is not always recognizable, owing to development of fat in the interstitial tissue. After some years, shoulders and arms are attacked; the change is one of pure atrophy, *i.e.* not complicated with fatty changes.

The following cases show that not only one, but at least two types of this etiological secondary form exist.

Zimmerlin gives seven cases, belonging to two families: four in one family (Loosli, Bern), and three brothers (Schuhmacher, Frickthal).

In the Loosli family ten children are living out of eleven. Two eldest, 28 and 30, are strong and healthy, also three youngest, 14–19. Of five between 20 and 26, one brother, 24, quite healthy. The other four, two brothers, 26 and 22, and two sisters, 25 and 20, are affected, and show severe disturbance of nutrition and function of voluntary muscles.

The father has pulmonary phthisis, mother quite healthy. No neurotic heredity. Family intelligent and well-built.

As an example, Verena Loosli, is 20 years old. Childhood weakly, with croupous pneumonia; at puberty she had erythema nodosum. Probable commencement of illness in summer 1881, with weakness in the back, fatigue in standing or sitting upright. In autumn, pain in region of right shoulder-blade, fatigue in both legs, with slight formication.

Since January, 1882, patient remarked that her shoulder-blades gradually began to come forwards, and the posterior borders projected from the chest-wall, chest flattened, clavicles more prominent, at the same time she had some weakness in the upper extremities; could not lift up her arm to her head, but could raise arms to the horizontal position by means of the deltoids. The spine, at the sixth dorsal vertebra, was slightly painful on pressure. Atrophy and paralysis in a high degree of both serrat.

antic. magn., with complete loss of excitability of muscles and their nerves for galvanic and faradic currents. Decided weakness and degeneration-reaction of both pectoral major; moderate but distinct weakness and commencing degeneration-reaction of acromial part of right trapezius; lessened capability, atrophy and degeneration-reaction of right and left biceps brachii. Weakness but not atrophy in flexors of right elbow. No wasting of deltoids, of the small muscles of hands, or interossei, no fibrillar tremor. Sensibility of skin normal.

Treatment consisted of methodical galvanisation of cervical spine, galvanisation and faradisation of affected muscles, moderate hydropathic course, and it produced some slight improvement.

Of the other members of the family, in two brothers aged 26 and 22, the disease began at 21 and 18 years respectively, while in a sister, aged 25, the first symptoms were noticed at the age of 23. One brother had a slight systolic apex murmur, and slight pulmonary phthisis. The disease had the same commencement, and the same general characters as in the case described.

Summary.—Identical points in all four cases are:

Beginning of illness soon after puberty, between 18 and 23. Localisation in upper part of body (especially upper half of trunk and upper extremities, whilst lower part of body was almost entirely free from disease), predilection for certain larger muscles (serrat. antic. magn., pectoralis, biceps, triceps brachii, extensors and supinators of forearm of both sides of body), immunity of muscles of thenar and hypo-thenar, interossei and deltoids, absence of fibrillar tremor in atrophied muscles, absence of secondary "lipomatosis luxurians," no particular disturbance of sensibility, or at least decided absence of hyperæsthesia and anæsthesia. The disease presents itself in all four cases in a gradually increasing atrophy and weakness of the muscles mentioned, and deserves the name of "progressive muscle atrophy," inasmuch as it successively attacked different muscles, which gradually progressed to the highest degree of atrophy. It agrees further with "progressive muscular atrophy," in so far as (at least in Case I.) the faradic and galvanic investigation of the attacked muscles and their accessory motor nerves has produced characteristic results (excitability of muscles and nerve retained for both currents, as long as the atrophy of the muscles had not become extreme; degeneration-reaction, to a moderate degree, of the individual atrophied muscles).

The disease differs from progressive muscular atrophy, in the

hereditary disposition, and in not affecting the small muscles of hand and interossei, also in the absence of fibrillar tremor. It agrees with Erb's progressive muscular atrophy of youth, which affects the larger muscles of shoulder and arm, lasts for some decades, and then remains stationary and presents no fibrillar tremor; but differs in the fact that, in Erb's cases, the large muscles of thigh and buttocks are attacked; there is no electric reaction of degeneration and no family predisposition. The described cases agree with one of Schulze's in most points, but in his case hereditary disposition is absent. They differ from the cases of Leyden and Moebius, where the lower extremities are affected, no "lipomotosis luxurians" is present, the male sex chiefly attacked, and childhood is affected first.

Zimmerlin refers to another family, named Schuhmacher, in which three brothers were affected; no hereditary neurosis, no insanity; family consisted of thirteen children; seven died young, cause unknown; three have atrophic paralysis. The disease began at 15, 13, and 8 years old respectively, with weakness and atrophy of both serrat. magn., rhomboids, pectoralis major of both sides, and to a less degree, biceps, triceps, supinators and extensors of forearm; while the deltoids, trapezius, infra- and supra-spinati, and small muscles of hand are not affected, and no fibrillar tremor. Differs from the Loosli family in all being boys, and so resembles Leyden's cases. In one case the mouth was a little drawn to the left, and he could not whistle. Some atrophy of left biceps, and to a less degree of left triceps, but their tendon reflexes are present; some atrophy and weakness of supinators and extensors of wrists; extension and abduction of right thumb weak. In the third case, which has existed for thirty-four years, both arms are wasted and weak, even the interossei and small muscles of hand; both thighs affected and almost powerless, extensors worse than flexors; both knees contracted, arthritis deformans in the right; no patellar tendon reflex; no "lipomatosis," no fibrillar tremor now. This last case began first in lower limbs, afterwards affecting the upper limbs, and fibrillar tremor was observed at first.

Dr. Zimmerlin thinks the disease in the two families shows an inherited tendency. In the Loosli family, severe muscular work may have been a cause, and the patients thought the disease came on at the time of life when they worked hardest.

He thinks that three possible pathological causes of the disease exist: 1. Myopathic change, primary disease of the muscles;

2. Neuropathic condition, multiple neuritis (Leyden); 3. Localisation in spinal cord, as a form of chronic poliomyelitis anterior. The second condition is negatived by absence of anæsthesia, and of marked electric changes. An autopsy can alone prove whether the disease is in the muscles or the spinal cord.

C. E. Beevor, M.D.

Dr. C. Eisenlohr (*Neurolog. Centralbl.* Feb. 15, 1884) describes a case of chronic spinal meningitis, with secondary degeneration of the spinal cord, probably of syphilitic origin.

Among the most important symptoms were paralysis and loss of sensation of the lower extremities, both more marked on the left side, with rigidity of the right leg. Also very obstinate constipation, incontinence of urine, and dull boring pains in both legs and in the rectum. History of syphilis eleven years ago. Present symptoms date from about one year ago. Temporary improvement under brisk mercurial treatment. Appearance of bedsores over the sacrum and great trochanters. Loss of sensibility confined to posterior and outer aspect of the thighs and calves, fairly good on anterior aspect. Hyperalgesia of dorsum of both feet. Plantar reflex weak on the right, absent in the left. Right leg much wasted. Patient died nearly two years after admission.

Post-mortem—was found great thickening of the pia mater, which was adherent to the dura, from the middle of the cauda equina to the level of the 10th dorsal nerve. Nerves of cauda soldered together. The thickening extended in a less degree to the mid-dorsal region. Nerve-fibres of cauda much degenerated. So also are the posterior roots of the whole lumbar region, more particularly on the right side, anterior roots having escaped.

Cord.—Lower sacral lumbar region, whole posterior columns quite degenerated. Higher up, however, the degenerated area becomes smaller and confined to the median column. The degeneration can be traced up into the cervical region affecting a diminishing area of the postero-median columns, till it is lost in the nuclei of the funiculi graciles. The fibres of the right sciatic nerve are greatly degenerated.

The author remarks that this case adds testimony to the views already expressed by Kahler and Pick, that the so-called Goll's columns are not distinct topographically from the neighbouring region, but are simply the longest centripetial continuations of the posterior roots, in this case supplying the lower extremities.

H. H. Tooth.